DISORDERS OF RESPIRATION AND CIRCULATION

VON NEUSSER

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CLINICAL TREATISES
on the
SYMPTOMATOLOGY *and* DIAGNOSIS
of DISORDERS *of* RESPIRATION *and* CIRCULATION

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PART II
BRADYCARDIA AND TACHYCARDIA
WITH BIBLIOGRAPHY

NEW YORK
E. B. TREAT & COMPANY
1908

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PREFACE TO AMERICAN EDITION

The modern work in laboratories upon infections has thrown into the background the consideration of disorders of many of the vital organs.

The heart as the *fons et origo* of the circulation must be seriously considered in practically every disease and its condition is many times the most reliable index of the result and best indicates the progress of the affection. Prof. v. Neusser has done well to bring together so ably all the factors involved in the decrease and increase of the cardiac action and to emphasize the fact that the study of the cardiac phases is no unimportant part of the work of every physician.

The edition in English has been amplified by the addition of abstracts upon and the bibliography of Bradycardia and Tachycardia, the work of Dr. Malcolm Douglas.

ANDREW MACFARLANE.

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PART I

BRADYCARDIA

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I

BRADYCARDIA

Conditions which Induce Bradycardia.—The conception of bradycardia, in the strict sense of the term, assumes that when each cardiac systole corresponds to a beat of the pulse, the frequency of the heart-beats is retarded. Cases in which there is only one palpable pulse-beat to every two cardiac systoles, of which one is not transmitted to the peripheral arteries, can hardly be regarded as true bradycardia; they belong preferably to the group of arhythmia and allorhythmia.

Experiment as well as observations at the sick-bed has shown that bradycardia may be produced (1) by excitation of the vagus center; (2) by irritation of the vagus at any point from its origin to its intracardiac ending; (3) by influencing the heart muscle itself. Experiment has also demonstrated the fact that the bradycardia of the first group disappears after section of the vagi or injection of atropin, which paralyzes the vagus terminals. That of the second group, due to excitation of the inhibitory vagus nerve anywhere along the entire length of the nerve, is overcome only by the action of atropin and not by vagotomy. The third group, the true cardio-muscular bradycardia, is not affected by either atropin or vagotomy. This cardio-muscular bradycardia may be the result (1) of impairment of the automatic excitability of the heart muscle from fatigue and exhaustion or from toxic influences; (2) of diminished conductivity of the heart muscle from defective nutrition in consequence of the influence of toxins, from excitation of the vagus and from block at the atrio-ventricular junction; (3), of weakened contractility of the heart muscle in consequence of anatomical lesions as myocarditis, fatty degeneration, coronary sclerosis and destructive poisons. While atropin may slightly increase the pulse frequency by paralyzing the vagus and thereby increasing the conductivity of the heart in the first and second groups of cases, it exercises no effect whatever on the third group.

Toxic Bradycardia.—At the bedside bradycardia is observed accompanying numerous affections. It is a frequent symptom in many intoxications, which will first be considered.

Nicotine.—The heart poisons play an important rôle, especially nicotine. Experimental pathology has demonstrated the fact that nicotine in small doses irritates the intracardiac vagus terminals and paralyzes them when the effect is continued. The increase of blood pressure resulting from irritation of the vasomotor center is, by paralyzing the latter, changed to a fall in the blood pressure. It is a peculiar fact, however, that bradycardia is rarely observed in men as a result of tobacco poisoning. Tachycardia with arhythmia usually occurs in chronic tobacco poisoning. The arhythmia is due to extra systoles, and the pulse as a result is generally small and soft. Retardation of the pulse appears as a symptom of those forms of tobacco poisoning which run the course of stenocardia, most probably caused by a spasm of the coronary arteries, and accompanied by retrosternal pain, dyspnœa, tremor of the extremities, together with vertigo and fainting spells.

The long continued misuse of tobacco may also lead to organic changes in the heart and vascular system. I have observed a case of myocarditis in an inveterate smoker characterized during life by irregularity and inefficiency of cardiac action, while the autopsy disclosed an enormous dilatation of the heart and atrophy of the papillary muscles, especially of the auricles, with fatty degeneration and atrophy of the musculature. Another large consumer of tobacco manifested, in addition to cyanosis and congested liver, a markedly visible swelling of the jugular veins, which did not pulsate. Simultaneous derangement of vision—scotoma for red and green, and considerable impairment of vision—were interpreted by Professor Bergmeister as a consequence of the overindulgence in tobacco. It was a striking fact in this case that, in spite of cyanosis and congestive manifestations, the second pulmonic sound was accentuated, although neither the left ventricle nor the lungs indicated any cause for the increased pressure. The conclusion readily suggests itself that the cardiac affection was here chiefly localized in the auricles, manifesting itself in their dilatation and diminished contractile power. The absence of pulsation in the dilated jugular veins is explained by the inefficiency of the right auricle, while that of the left auricle led to increased pressure in the pulmonary system. The signs of dilated right or left ventricles were absent; cardiac dullness was barely increased, and the cardiac impulse not displaced. Marked improvement followed the complete abstinence from tobacco. These observations force upon us the reflection whether the deleterious effect of tobacco affects first the auricles. It would be interesting, when analyzing the pulse curves in chronic nicotine poisoning, to note whether the intermissions occasioned by the extra systoles originate from the auricles or ventricles.

Lead.—Bradycardia is not infrequent in chronic lead poisoning, especially in the clinical picture of lead colic. The slow pulse is a prominent symptom in the coma of encephalopathia saturnina. I recollect from the time of my assistantship a case in which there was delirium, rigidity of the neck, and retracted abdomen, and which was wrongly diagnosticated in the psychiatric clinic as meningitis. Tachycardia may occur as a manifestation of vagus paralysis when the poison causes a vagoneuritis. A painter, exhibiting cerebral symptoms which were interpreted as encephalopathia saturnina, died from pneumonia, with tachycardia and terminal hematemesis. The autopsy disclosed neuritic degeneration of the left vagus, thus proving that tachycardia may also develop on this basis.

Digitalis; Strophanthus.—Digitalis is another poison productive of bradycardia, and to this beneficent property it owes its reputation as a curative agent. There are, however, cases of chronic digitalis poisoning, especially among military conscripts, who take digitalis for a considerable time in order to be rejected from service. Under such circumstances the diagnosis of chronic digitalis poisoning may be difficult. The diminished frequency of the pulse may, through cumulative action, be changed into the opposite condition and an arhythmia of the heart may occupy the foreground. Simultaneously there is disturbed digestion, coated tongue, vomiting, insomnia, cerebral symptoms, cephalalgia, and sometimes diplopia. The pulse becomes small and frequent, and death sometimes ensues.

Chromatopsia has been observed to be an important manifestation of digitalis poisoning. There may be blindness for green, and such patients see the landscape in the spring and summer as if covered with snow. A general with heart disease, who took overdoses of digitalis, experienced serious bradycardia and violet vision. His principal complaint was that his lamp burned badly with a violet flame. In poisoning it is therefore important to keep in mind these concomitant manifestations of the effect of digitalis.

The action of digitalis upon the circulatory apparatus and its nerve supply is very complicated. At first it irritates the pneumogastric, and may therefore indirectly cause retardation of the heart-beat by inhibition. Digitalis also increases the tonus of the heart muscle and the contractions of the heart are stronger. In large doses, however, this irritation of the heart muscle may cause extra systoles. Finally, digitalis contracts the vessels, and may thereby lead to increased pressure in the heart and vessels. The principal therapeutic effect of digitalis is chiefly manifested by increased systolic contractions of the heart and interference with conductivity through irritation of the pneumogastric. Its vaso-constrictor effects, however, may also play a rôle in bradycardia. The increased blood pressure leads to an irritation of the vagus nucleus and transmission of the same to the intracardial vagus ends, thereby diminishing the conductivity still more.

Other poisons employed in the treatment of cardiac affections, as strophanthus, may act analogously to digitalis. A druggist's assistant, wishing to take some brandy, took by mistake a bottle containing tincture of strophanthus. The symptom complex consisted in unconsciousness, coldness of the extremities, reduction of the pulse frequency to 40; the patient recovered.

Physostigmin; Muscarin.—The list of poisons producing bradycardia includes physostigmin, which irritates the intracardial peripheral vagus ends, and muscarin, which apparently is able to produce bradycardia by an inhibitory effect upon the intracardiac apparatus. Physostigmin is frequently employed as a purgative in veterinary practice, and Von Noorden recommends the same, physostigmin-salicyilate, in the meteorism of typhoid. In animal experiments pulse retardation due to physostigmin or muscarin is counteracted by atropin, which paralyzes the vagus ends, but is not affected by vagatomy. Retardation of the pulse in muscarin poisoning results from irritation of the intracardiac inhibitory end apparatus, or from diminished contractility of the heart, especially when large doses of the poison have been taken. In the former case bradycardia is overcome by atropin. but not in the latter. This is observed in poisoning with fly agaric.

Potash Salts; Picric Acid.—Destructive poisons like potash salts and picric acid induce bradycardia by injuring the heart muscle. In a case of picric acid intoxication, bradycardia and fainting were accompanied by the well-known picric icterus, and the acid could be demonstrated in the urine.

AUTO-TOXIC BRADYCARDIA—*Ptomaine Poisoning.*—The question as to whether true bradycardia occurs in ptomaine poisoning has not been clinically decided. Janowsky mentions bradycardia as a resultant of sausage poisoning (botulism). In the toxicological experiments with the toxins of the bacillus botulinus (Von Ermengen), bradycardia was not observed, by its action on the vagus or its terminals. The clinical manifestations of botulism are exceedingly varied: dryness of the throat, paralysis of the ocular muscles, typhoid and choleriform symptoms, while retardation of the pulse occurs only exceptionally, and then only terminally, and can be well explained by injury to the heart muscle.

Generally speaking, the symptomatology of ptomaine poisoning has been but slightly differentiated from a clinical point of view, owing to the consideration due to bacterio-infectious elements as well as to the toxic, The isolated cases of bradycardia due to sausage poisoning scattered through the literature are probably to be regarded from the same point of view as the cases of bradycardia in phosphorus poisoning reported by French physicians. It should not be lost sight of that they may have been caused by special complications, such as hemorrhagias into the sheath of the vagus or direct injury to the heart muscle owing to special localization in the heart. In the many cases of phosphorus poisoning we have never observed bradycardia, but often a small, soft, and frequent pulse.

Bile.—Bradycardia is also caused by autogenous poisons resorbed by the organism, similarly to intoxications resulting from ingested poisons. The principal representative of this group is the icterus caused by biliary engorgement, in which retardation of the pulse occurs through retention of the constituents of the bile, especially the biliary salts. It may result from chemical irritation of the vagus center, from excitation of the peripheral intracardiac vagus terminals, or also from the continuous toxic effect through direct injury to the heart muscle. That the vagus is the real point of attack of the bile acids is proved by the fact that bradycardia in icterus is removed by injections of atropin (Weintraud). Atropin naturally will have no effect in those cases where the bile acids have caused bradycardia through anatomical lesions of the heart muscle.

In polycholic icterus, where bile flows freely into the intestine and the stools are colored dark green, there is no bradycardia. This explains why in biliary hypertrophic cirrhosis, even in the rare cases where there is temporary acholia owing to transient occlusion of the biliary ducts, there is no retardation of pulse. This negative phenomenon may under certain circumstances serve as a guide to the differential diagnosis from protracted icterus catarrhalis with permanent biliary obstrúction, in which there is sometimes a considerable enlargement and induration of the liver and spleen. Icterus catarrhalis, even when protracted, is in the majority of cases a curable disease, while biliary hypertrophic cirrhosis is, without exception, fatal. Bradycardia in icterus is therefore a favorable prognostic symptom, provided it is not caused by direct injury to the heart muscle or by a cerebral complication, as pachymeningitis hemorrhagica.

Uræmia.—As in icterus, bradycardia may occur in uræmia, another auto-intoxication, and here also may have a prognostic significance. A perceptible retardation of the pulse in the convalescence of scarlet fever in youth should arouse attention. In the course of scarlet fever, tachycardia is one of the most important clinical signs, but the sudden onset of bradycardia is not infrequently the first symptom to announce the onset of a nephritis, with uræmia. Pulsus rarus et durus in scarlet fever, the result of toxic irritation of the vagus center and increased nephritic pressure, is therefore an ominous sign, whereas bradycardia with pulsus mollis in the course of convalescence from scarlet fever should be regarded as favorable a symptom as it is in other infectious diseases which will be considered later.

In elderly people suffering from nephritis, retardation of the pulse may very well be a symptom of uræmia or a sign of a cerebral hemorrhage complicating the nephritis.

Acetic Acid; Acetone.—Auto-intoxications in which bradycardia is of importance as a clinical phenomenon, and is often misinterpreted, include those forms in which acetic acid and acetone occur in the urine. A patient of mine presented the following symptoms: Coated tongue, vomiting, rigidity of neck, retracted abdomen, and strikingly retarded pulse. Meningitis was the first thought. Examination of the urine showed a large quantity of acetone. Calomel was administered, the meningitic symptoms disappeared, and recovery followed. Bradycardia, however, is not present in all cases in which examinations of the urine demonstrate large quantities of acetone and acetic acid. Two cases of hydrophobia which I had an opportunity to follow clinically, and which presented marked acetonuria, manifested an acceleration of pulse—no bradycardia. Also in coma diabeticum, in which diaceturia and acetonuria are present, the pulse has been found retarded by other observers. The cases observed by me showed a remarkably soft and frequent pulse until death. It would therefore seem that in all auto-intoxications it depends upon the quantity of poison, together with other factors, as to whether an irritation or paralysis of the pneumogastric results.

Convalescence from Acute Infectious Diseases.—Toxic bradycardia also includes those forms which occur in the convalescence after acute infectious diseases, as pneumonia, erysipelas, typhoid, rheumatism, measles, diphtheria, and scarlet fever. In the course of diphtheria retardation of the pulse may be of a diverse significance. The transitory form of bradycardia, which occurs in the convalescence after diphtheria, without other complications on the part of the heart or the nerve centers, and is frequently accompanied by polyuria, belongs clinically to the group of post-infectious bradycardias. In diphtheria, bradycardia may also be of unfavorable prognostic significance in consequence of myocarditic degeneration, involvement of the pneumogastric, or finally direct invasion of the bacillus diphtheriæ into the oblongata. This last cause was positively established in one patient of Barbier. In the severe and complicated cases of diphtheria, accompanied by paralysis of the soft palate, tachycardia with arhythmia usually develops in consequence of involvement of the vagus and of the myocarditis. Both bradycardia and tachycardia with simultaneous arhythmia, and accompanied by considerable anorexia, pallor of the face and diaphoresis are not rarely premonitory signs of fatal syncope.

The rapidity and weakness of the pulse are in the great majority of cases of diphtheria to be regarded as the direct consequence of the intoxication and disappear after the injection of anti-diphtheritic serum. The persistence of tachycardia in spite of the serum treatment is characteristic of severe cases, and is, according to Barbier, an urgent indication for an augmented dose of serum. The reappearance of an accelerated pulse in diphtheria patients treated with serum is likewise a sign of the recrudescence of the disease (Barbier).

Bradycardia is very often noted in the decline of acute articular rheumatism, especially in mild cases not complicated by cardiac involvement. Retardation of the pulse is a well-known occurrence in gonorrheal polyarthritis. In complications with pericarditis or endocarditis the pulse frequency usually rises. The persistence of the tachycardia after the disappearance of the articular signs should always give rise to the suspicion that the endocardial process has not yet ceased, especially if the tongue is coated and there are occasional rises of temperature.

The following may be cited as an example:

A young girl entered the clinic of the General Hospital, suffering from rheumatism and endocarditis. Her condition improved, the mitral insufficiency became stationary, and, aside from occasional slight rises in temperature and accelerated pulse in the evening, she had no discomfort. As she felt otherwise well, she was at her own request permitted to leave the hospital. Hardly had she stepped into the courtyard of the hospital when she suddenly collapsed and had to be brought back to the hospital, where she died shortly afterward. The autopsy disclosed a fresh embolus of the arteria fossæ Sylvii and a mitral endocarditis which had not become quiescent. There are, however, exceptions to this general rule, and a slow pulse may be found in the course of rheumatic endocarditis in spite of fever and dyspnœa. A patient in our clinic with endocarditis following articular rheumatism had a pulse constantly of 60 per minute, temperature 38 degrees, and respirations 30. The patient also suffered from diarrhœa and colicky pains.

Another affection in which bradycardia may be of considerable diagnostic and prognostic significance is influenza. There are cases in which the tachycardia may become as pronounced as in experiments with vagotomy. Cases of la grippe also occur with a pulse frequence of 48-44 per minute, as described by Le Clerc and Barthelemy. At the Wiesbaden Congress Vogel presented a report on the influenza epidemic of the Munich garrison: in 46 per cent. of the cases there was bradycardia, a surprising percentage, the cause of which may perhaps be connected with local conditions (beer heart ?). Sometimes after recovery bradycardia is frequently noted with a strikingly soft pulse, and this is often looked upon as a criterion for the complete recovery from influenza.

In some cases bradycardia changes permanently into tachycardia. Pulse retardation may therefore assume considerable significance in diagnosis and prognosis during an influenzal epidemic, for the following reasons: (1) It may be a sign that influenza really has preceded; (2), it may be a warning that recovery has not yet completely taken place, and that patients are subject to relapse and the sequelæ of influenza. The bradycardia in influenza is probably due to a direct injury of the heart muscle.

Causes of Toxic Bradycardia.—The causes of the pulse retardation already mentioned may be various. The poisons or the toxins developed in the organism may either have a direct effect upon the heart or cause bradycardia by vagus irritation. A cardinal difference between the intra- and extra-cardial forms of bradycardia lies in their behavior toward atropin. The extra-cardial retardation, occurring through excitation of the vagus center or of the vagus anywhere to its termination in the heart, is counteracted by atropin, whose paralyzing action upon the vagus terminals has already been noted, while those forms of bradycardia occasioned from diminished contractility of the heart are indifferent to atropin (Dehio).

The investigations by Engelmann and his pupils have shown that regular cardiac activity consists of three principal functions of the cells of the heart muscle: (1) automatic irritability; (2) conductivity, that is, the power to transmit motor irritations from one muscle cell to another, and (3) contractility of the muscle cells. The vagus and accelerans do not act upon the heart muscle, causing independent contractions, as does, for instance, the crural nerve on the quadriceps. They rather influence either in a negative or positive sense the conductility and contractility of the heart muscle, the vagus irritation affecting at first the auricles. The functional disturbances of the heart resulting from nervous influences are in general variable, while those caused by diminished conductility of the heart muscle are usually constant. Bradycardias, therefore, which have persisted unchanged for a number of weeks or months may almost with certainty be looked upon as of the true cardiac form. Retardation of the pulse to one-half the normal frequency, as occurs in the convalescence from typhoid, influenza, and other infectious diseases, is caused by reduced conductility of the heart muscle; that is, the inability to transmit the contractile irritation arising at the venous orifices. Owing to this decreased conductility there may be impairment of ventricular contraction, and also of the auricular contraction, if the inhibition of the power of transmission continues. Since the conduction at the atrio-ventricular junction meets with difficulties even under physiological conditions, interference with transmission may lead to bradycardia by the fact that only every second or third irritation is conveyed to the ventricles, the auricles all the while pulsating regularly. This is the so-called heart-block, which means inhibited transmission of irritation at the atrio-ventricular junction, which clinically manifests itself by pronounced bradycardia of the ventricles. That the auricles in many cases continue to pulsate more often is proved not only by the X-ray picture, but also by the fact that the jugular veins pulsate more frequently than the apex of the heart and the radials. These forms of bradycardia based upon weakened intracardiac conductility are, however, not independent of nervous influences. The conductility may be inhibited by vagus irritation, and the bradycardia thus intensified, as is the case in the acute attacks of Stokes-Adams disease.

CAUSE OF THE BRADYCARDIA IN STOKES-ADAMS DISEASE.—The cause of the paroxysmal retardations of the pulse in Stokes-Adams disease, associated with periodical attacks of unconsciousness, is to be found—as demonstrated in the majority of the observed cases—partly in lesions of the medulla and partly in simultaneous affections of the heart itself.

About two years ago a typical case of this affection was observed in our clinic. There was fatty degeneration of the heart, confirmed by autopsy, due to sclerosis of the coronary arteries and atheroma of the basilar artery. During the paroxysms the pulse was reduced to 30. The cause of this bradycardia was here evidently the defective contraction and the diminished conductivity of the heart, depleting the aorta and the vessels branching from it, and therefore also the oblongata. Besides, the oblongata was originally poorly supplied with blood on account of the affected basilar artery. In this patient, therefore, there was a complete vicious circle in the etiology of symptoms. The bradycardia caused by the coronary sclerosis—bradycardia of conduction—is augmented during the paroxysms through the irritation of the vagus center from the ischemia of the oblongata. The vagus irritation, however, serves to still further impair the conductivity, the aorta and vessels are still more poorly supplied, a deficiency which again acts upon the oblongata and vice versa. The irritation of the vagus inhibits the cardiac conductility and the retardation of the pulse caused thereby may still further be intensified. This pernicious effect of increased irritation of the vagus was strikingly illustrated in the above mentioned case after the administration of digitalis, which was given to the patient on account of a coincident arhythmia. The pulse sank to 16 beats a minute, and there was deep coma from which the patient never awoke. This case serves to confirm Wenckebach's claim that in cases of arhythmia which result from the inhibition of conductivity, digitalis is a dangerous remedy, just because it irritates in the first place the pneumogastric, and thereby inhibits the conductivity still more. If, then, these observations go to prove that bradycardia caused by inhibition of conductivity may be still further increased by vagus irritation, it is clear that by the exclusion of the vagus tonus, the conductivity may be augmented. Atropin can in these cases of retarded pulse increase the frequency of the pulse, although not so markedly as in extracardiac bradycardia which has been occasioned by vagus irritation, where the cardiac conductivity is intact. The above cited dictum of Dehio, advocating the administration of atropin for the determination of a differential diagnosis between intra- and extracardiac bradycardia, cannot be taken dogmatically. Atropin may bring about an acceleration of the pulse by eliminating the vagus tonus, even in intracardiac bradycardia resulting from reduced conductivity of the heart muscle, regardless of whether the same is produced by defective nutrition of the latter by poisons (typhoid, influenza, diphtheria toxins), or finally by obstruction at the atrio-ventricular junction. On the other hand, those forms of bradycardia which result from impaired contractility, and in which the weak contractions of the heart muscle do not cause any pulsation at the periphery, do not react to atropin.

Bradycardia in Affections of the Heart and the Coronary Arteries.—These bradycardias occur in a number of cardiac affections in which there are anatomical lesions of the heart muscle, as fatty degeneration, coronary sclerosis, and toxic myocarditis. In arterio-sclerosis there is retardation of the pulse and *pulsus rarus et durus* is not uncommon. The heart, so to speak, fights with the peripheral circulatory apparatus and endeavors to prolong diastole as much as possible, in order to overcome the resistance and to exert the maximum of systolic power after the diastole. In other cases the pulse retardation is traceable to a direct impairment of the heart muscle through arterio-sclerosis, as, for instance, in the paroxysmal bradycardiac attacks with loss of consciousness of Stokes-Adams disease, or in the true stenocardias caused by disorders of the coronary vessels.

To this group of bradycardias which depend on the heart and circulatory apparatus may also properly be classed those which occur with ague, coldness of the skin, vertigo, dilatation of the pupils, unusual hardness and retardation of the pulse, with accentuation of the second aortic sound and complicated by an acute dilatation of the heart. Jacob explains these paroxysms by the contraction of the peripheral vessels produced by irritation of the sympathicus, and describes an angiospastic bradycardia.

The most pronounced retardation of the pulse down to 18 and 16 beats per minute is especially observed in thrombosis and embolism of the coronary arteries. The pulse becomes progressively slower, and as the bradycardia increases, death may ensue. The persistency and constant augmentation of the pulse retardation characterize just these cardiac forms of bradycardia, while those which depend on irritation of the bulbar vagus center or its cardiac branches disappear ante mortem. Excitation of the vagus is followed terminally by a vagus paralysis: a phenomenon also observable in meningitis, to which reference will be made later on.

Bradycardia in the course of coronary angina is a serious symptom. The ischemia of the myocardium causes impairment of the cardiac transmission of stimuli, and the pulse retardation is due to this absence of ventricular contraction. In the well-known animal experiment of Cohnheim, with the coronary arteries clamped off, the rhythmic contractions of the auricles outlasted those of the ventricles, so that the former constitute the ultimum moriens. This is confirmed in man by the following case. When an assistant to Bamberger, I was called one night by a nurse to sign the certificate of death of a patient suffering from a heart affection, who had suddenly died. I had demonstrated this case the same afternoon as atheroma of the aorta with valvular insufficiency. As there were also stenocardiac manifestations, I made a tentative diagnosis of atheroma of the coronary arteries. On inspection of the body I found no pulse, the pallor of death, no cardiac sounds; in fact, cessation of cardiac function. To my surprise, however, I observed pulsations in the veins of the neck, then showing the continuance of auricular contractions. There occurred a respiratory gasp, the venous pulsation ceased, and all was quiet. At autopsy the diagnosis of occlusion of the coronary arteries was confirmed.

In mitral stenosis a slow pulse is the exception, even in cases where no digitalis had been administered. This resembles the bradycardia of arterio-sclerosis, where the left ventricle prolongs its diastole in order to collect as much blood as possible for the succeeding systole. There seems to be a spontaneous bradycardia, a form of auto-correction of the disturbed circulation in serious affections of the orifices. In mitral stenosis, with degeneration of the heart muscle there is also an apparent retardation of the pulse when the contraction of the left ventricle is so weak that it can no longer produce peripheral pulsation. In this case the number of radial pulsations corresponds to twice the number of cardiac contractions, a fact which is demonstrable by inspection and palpation of the cardiac impulse and by auscultation. In many cases, however, of this apparent retardation of the pulse, extra systoles play a part. Bradycardia is much more frequent in stenosis of the aortic orifice.

Of great prognostic significance are those forms of cardiac bradycardia which occur not only after the crisis of, but even in the course of pneumonia. When there is a striking retardation of the pulse in the course of an inflammation of the lungs, a complication on the part of the brain, especially a meningitis, should at once be thought of. There are, however, cases of retarded pulse with complete consciousness, in which there is neither cephalalgia, vomiting, nor any other meningeal manifestation, and which justify the assumption of irritation of the vagus center. They are those rare cases of bradycardia in patients with pneumonia, suffering from fatty heart, which may lead to great prognostic errors, especially at the time of the crisis.

An old man, suffering from pneumonia, exhibited at the time of the crisis subjective euphoria and great retardation of pulse. On superficial observation these two symptoms might have given rise to a favorable prognosis, but edema of the ankles, which soon appeared, put the bradycardia in a different light. The retardation of the pulse continued, and death ensued with manifestations of pulmonary edema. Autopsy showed pneumonia in the stage of resolution, edema of the lungs and brain, and fatty heart. Bradycardia, therefore, in the course of infectious diseases and during convalescence may have different causes and may be the first sign of a latent fatty heart, as shown by this case.

Sendler observed bradycardia following compression of the pulmonary artery by a fibroma, and explains it by defective supply to the coronary arteries in consequence of the diminished blood stream to the lungs and the left heart.

BRADYCARDIA IN DISEASES OF THE BRAIN.—Bradycardia occasioned by excitation of the vagus center and its intracardiac terminals may have manifold causes: anatomical lesions of the oblongata, hyperemia of the vagus nucleus—increasing the blood pressure on one side and irritating the vagus by congested blood on the other—sudden ischemia of the oblongata, chemical irritation of the vagus center in intoxications and auto-intoxications, and finally reflex irritation of the branches of the vagus transmitted to the vagus center from other nerves; for instance, the trigeminus.

In this way bradycardia occurs as a symptom of various affections of the brain. The diagnostic importance of a slow pulse in inflammation of the meninges is well known; it is always present in tuberculous and serous meningitis, less so in the cerebro-spinal variety and in the forms of meningitis caused by pneumococcus or streptococcus infection.

Following the retardation of pulse in tuberculous meningitis there will be paralysis of the vagus center and tachycardia in the stage of increased exudation at the base of the cerebrum. This change from bradycardia to tachycardia is an important differential diagnostic sign of meningitis.

Bradycardias occurring in affections of the ear and due to the presence of foreign bodies in the external ear are to be attributed to reflex irritation from the auricular branch of the vagus. In concussion of the brain and cerebral hemorrhages the slow pulse is one of the cardinal symptoms, and the pulsus cephalicus in cerebral apoplexy has long been known. Cerebral hemorrhages are distinguished from the apoplectiform attacks of disseminated sclerosis by the absence of bradycardia in the latter and the presence rather of tachycardia, which may probably be explained by reason of the cerebral sclerosis not permitting increased pressure in the brain. From a prognostic point of view, bradycardia in cerebral hemorrhage is a much more favorable symptom than tachycardia, because the latter nearly always indicates a fatal termination when accompanied by high temperature.

In various cerebral conditions the retarded pulse results partly from direct and partly from indirect action upon the vagus center. The indirect effects occur in acute increase of the cerebral pressure, as in rapidly growing tumors, in acute hydrocephalus, hemorrhage into the ventricles, hematoma of the dura mater, or in meningitis, which also acts by reflex action through irritation of the dural branches of the trigeminus. Slowly growing tumors like gliomata may develop without producing bradycardia, as the vagus nucleus accommodates itself in time to the slowly increasing pressure. A direct effect may be exerted upon the vagus center by the sudden onset of anemia of the oblongata, such as may occur in atheroma of the basilar artery, also by a sudden ischemia of the latter from compression of the carotid, or obstruction of the carotid by an aneurism of the aorta.

Pulse retardation to 44 beats has been observed in softening of the occipital lobe consequent upon embolism of the arteria profunda cerebri. In a case of Peacock, of stenosis of the foramen occipitale magnum owing to ossification between the occiput, atlas and alveolar process, following injury to the upper cervical region, the pulse was usually 32, sinking to 12 and 7 beats during periods of unconsciousness.

In hemorrhages of the fourth ventricle, in polioencephalitis acuta hemorrhagica ant. adjacent to that part of the vagus nucleus which lies dorsal and lateral to the hypoglossus nucleus, there may occur, varying with the severity of the lesion, irritation of the vagus nucleus with bradycardia, or tachycardia if there is paralysis. In tumors of the fourth ventricle bradycardia is not an infrequent manifestation, accompanied by vomiting, paralysis of the ocular muscles, abducens and facial paralysis, less often paralysis of the oculomotorius, acusticus and glosso-pharyngeus. A slow pulse occurring simultaneously with paralysis of the facialis, and later associated in its further course with neuralgia of the superior branch of the trigeminus, vertigo, agoraphobia, and finally paralysis of the masseters, has been described in tubercle of the oblongata. Paralysis of the facialis, acusticus, and glossopharyngeus with simultaneous retardation of pulse, choked disk, and wavering gait, have been observed in gummata of the pedunculus cerebelli med. The bradycardia was here the consequence of a lesion of the vagus trunk just outside the oblongata.

In cysticerci of the fourth ventricle, bradycardia frequently occurs as a concomitant manifestation with vomiting, vertigo, intermittent occipital and nuchal cephalalgia, nystagmus, facial anesthesia, unstable gait, and glycosuria. I have myself seen such a case in a young girl who entered the hospital with the diagnosis of hysteria, and suddenly died in convulsions with a slow pulse. Autopsy revealed a cysticercus of the fourth ventricle the size of a cherry. Bradycardia, therefore, which occurs with vertigo and epileptiform paroxysms should call attention to the oblongata, while, strange as it may seem, bradycardia has not so far been observed in bulbar paralysis accompanied by atrophy of the nerve nuclei. It has been found, however, in the bulbar form of tabes and syringomyelia.

BRADYCARDIA IN STOKES-ADAMS' DISEASE.—In Stokes-Adams' disease also, which is characterized by permanent retardation of pulse, as well as by frequent epileptiform and apoplectiform paroxysms, the irritation of the vagus nucleus plays a part. The anatomical substratum of this disease is in many cases an arteriosclerosis of the coronary vessels, of the basilar artery, of the vertebral arteries, and the arteria nutritia vagi. There are, however, cases which present clinically the picture of Stokes-Adams' disease which, at autopsy, show neither arterio-sclerosis nor arteritis due to any other cause (syphilis, infectious diseases, etc.), also cases in which the cerebral and coronary arteries are only slightly affected, and finally cases in which—like in Brissaud's case—a gumma of the middle crus cerebelli produces during life the symptom-complex of Stokes-Adams' disease. The Stokes-Adams' disease is, therefore, not an entity, neither in regard to symptoms observed at the bedside, nor to the findings established at autopsy. The typical cases, which are accompanied by apoplectiform or epileptiform paroxysms, must be differentiated from epilepsy and apoplexy. The apoplectiform paroxysms in Stokes-Adams' disease run their course without paralysis of the extremities and cerebral nerves. It is therefore necessary for a differential diagnosis to make use of all symptoms of value for the diagnosis of organic diffuse affections, as Babinsky's phenomenon, conjugate deviation, etc. The very persistence of bradycardia in epileptiform paroxysms without the cry or biting of the tongue differentiates these forms of bradycardia from the transitory ones of genuine epilepsy.

In essential epilepsy, retardation of the pulse occurs rarely, except perhaps in cases with simultaneous hydrocephalus. Possibly Napoleon belonged to this class of epileptic bradycardia, as, according to Corvissart, he had a habitually slow pulse down to 40. During the last years of his life, when Napoleon suffered from cancer of the stomach, and from September, 1819, while under the observation of Autommarchi, his pulse was small but regular at 60 per minute; later it rose to 70-80, reaching even to 90 in April, 1821. Napoleon is said to have been epileptic, was of small stature, but had a cranial circumference of 56.4 cm., and a small heart. This point should be emphasized, because, according to Jastrowitz, not only may cardiac hypertrophy occur in epileptics, but bradycardia in epilepsy may even be of cardiac origin. Bradycardia occurs also in diabetes mellitus without demonstrable lesion of the brain and circulatory apparatus. Such a case I have seen only recently. In Frerichs' case there was simultaneous paralysis of the facialis, "pointing to an affection of the fourth ventricle. The pulse repeatedly went as low as 40, and had at one time the characteristics of the pulsus bigeminus."

BRADYCARDIA IN HYSTERIA, MELANCHOLIA, BASEDOW'S DISEASE.—Von Noorden, in the Charity Annals of 1893, described some interesting cases of bradycardia in hysterical individuals. The hysterical nature of the bradycardia manifested itself in the accompanying symptoms, the hyperesthesia and anesthesia of the fauces and glottis, extending often into the larynx itself, and even into the trachea, the vomiting, and the influence of suggestion upon these manifestations. In melancholia the pulse in the majority of cases is regular, small, and infrequent; in melancholic frenzy it is tense, and also slow. Opium removes both tension and slowness. In morphinism the pulse is usually accelerated; in progressive paralysis there is sometimes bradycardia. Eichhorst has seen cases of Basedow's disease with temporary retardation of pulse, and the same was also observed in the transition stage from Basedow's disease to myxedema. Zülzer describes a bradycardiac vagus neurosis, the principal symptoms of which are distention of the lungs, slow pulse to 52, sensitiveness of vagus to pressure, thoracic oppression, sensation of fright, dyspnoea, and disappearance of the symptoms after the administration of atropin.

Bradycardia from Direct Irritation of the Vagus.—The cases of bradycardia which occur from direct irritation of the vagus trunk are numerous. Experiments by Czermak, Quincke, and Tanhofer show that pressure in the pneumogastric region of the neck may produce retardation of pulse. It is difficult to prove that this is caused by direct pressure upon the centrifugal fibers of the vagus, since the vagus also contains centripetal fibers, through which a reflex irritation of the vagus center may have been occasioned. This double function of the vagus seems to be of importance in disorders of the heart. How is it that the most pronounced cases of arhythmia are hardly perceived by patients, who are able to follow their sometimes arduous callings without experiencing oppression, while in other cases a slight arhythmia of short duration causes marked sensations of fright? I am inclined to believe that this sensation of fright occurs in those cases where the vagus excitation is transmitted centripetally to the oblongata. Fear would therefore depend upon a misuse of the double function of the vagus. Disorders of the cardiac function which are occasioned through centrifugal transmission to the intracardiac vagus terminals are only perceived when they cause secondary disturbances in the circulation. Centripetal irritation causes a sensation of fright by excitation of the vagus nucleus and transmission of the same to the neighboring nucleus of the acusticus situated in the oblongata, as well as to the respiratory and vasomotor centers. In a case of tabes with grave gastric crises and complicated with tetany, Hoffmann describes bradycardia of 30 pulse beats per minute, and explains this phenomenon by increased excitability of the vagus in consequence of tetany. Personally I have observed retardation of pulse with later terminal tachycardia in carcinomatous glandular metastasis of the foramen jugulare vagi. Bradycardia with dyspnoea, frequently alternating with tachycardia and vagus asthma, may occur as clinical symptoms in neuritic lesions of the vagus, in compression of this nerve by tuberculous or carcinomatous glands in the mediastinum, by tumors, and also by aneruism of the aorta.

The retardation of the pulse in carcinoma of the esophagus is explained by its anatomico-topographical relation to the vagus. In some cases the cause may be inanition, owing to stricture of the esophagus, in a similar way as it occurs in professional fasters.

BRADYCARDIA IN CARCINOMA OF THE STOMACH, GASTRIC ULCER, AND GALL STONE COLIC.—In carcinoma of the stomach the slow pulse may sometimes be of diagnostic importance, as an early symptom at a period when the tumor is not as yet demonstrable.

Bradycardia occurring in a person of advanced age, who previously had always had a good stomach, and is suddenly taken ill with symptoms of dyspepsia and cachexia, should therefore suggest the possibility of a latent carcinoma of the stomach.

Vagus irritation may manifest itself by other signs in addition to bradycardia, as sensitiveness to pressure of the vagus trunk in the neck, and ravenous appetite, as described by Ortner. Retardation of the pulse may also occur in other gastric affections, as dilatation of the stomach, dyspeptic chlorosis, gastric ulcer, but will disappear with improvement of the gastric disorder.

Bradycardia may be produced in animals by inflating the stomach, the explanation of which is apparently the reflex irritation to the vagus trunk. In gastric ulcer pulse retardation may outlast both gastric pain and vomiting, and may therefore be of diagnostic and prognostic importance as a sign of latent or still existing gastric ulcer. The slow pulse following gall stone colic may lead to the erroneous assumption of a grave coronary angina, especially if the pains radiate toward the cardiac region and the left arm. Thus I have observed a case where the pulse frequency was 45 after a paroxysm of epigastric pains radiating toward the left arm, and which the attending physicians diagnosed as a true coronary angina. The passage of gall stones after Carlsbad treatment disclosed the trouble to have been cholelithiasis. In helminthiasis, too, bradycardia may be expected now and then. I have, however, no personal observations to that effect, although other symptoms that should be regarded as vagus irritations, as paroxysmal vagus asthma, have to my knowledge been observed to occur with taenia. In meteorism following intestinal stenosis caused by cancer of the intestine, I observed distinct bradycardia, which was explained by reflex irritation of the intestinal branches of the vagus. Pulse retardations which accompany intestinal colics should be regarded from the same point of view, in contradistinction to the acceleration of pulse which is nearly always associated with peritonitic pains. I do not remember the occurrence of pulse retardation with abdominal pains in which a necropsy or biopsy at operation disclosed peritoneal adhesions. The peritoneum usually responds to all irritations with tachycardia. Peritonitis with bradycardia does not exist.

Transitory bradycardia may also be produced by reflex irritation of the vagus branches, especially excitation of the superior laryngeal. Pulse retardations in laryngoscopic manipulations, in faulty deglutition, and in tracheotomy is explained in the same way.

Bradycardia is also occasioned by reflex excitation of the cardiac inhibitory center on the part of other sensitive nerves, such as the trigeminus in neuralgia, in sciatica, appendicitis, and extensive dilatation of the bladder.

BRADYCARDIA IN INJURIES TO THE SPINAL CORD.—Finally, mention should be made of the slow pulse which has been observed in injuries to the spinal cord after fractures in the region of the fifth and sixth cervical vertebræ (dilatation of the pupil, pulse 56-48 in a child). Wallenberg describes a remarkable bradycardia (42) in a stab wound of the left third dorsal nerve, near the spinal ganglion, in a man who had several stab wounds in the left side of the back above the scapula. One of the stabs close to the spinal cord involved the third thoracic nerve. He explained the retardation of the pulse by section of the sympathicus branches of the third dorsal nerve between the visceral parts.

PROGNOSIS AND TREATMENT OF BRADYCARDIA.—The prognosis, and likewise the treatment, of the different forms of bradycardia are very variable, as seems natural from the preceding explanations. Gumma of the brain and myocarditis gummosa will require different treatment from Stokes-Adams' disease, which rests on an arterio-sclerotic basis and ischæmia of the oblongata. In the epileptiform paroxysms and the attacks of unconsciousness of this disease, amyl nitrit or nitroglycerin would be indicated, while potassium bromid would not be well borne. In bradycardial neurosis atropin is probably the indicated remedy. This is also true for those cases of Stokes-Adams' disease where the vagus components play a prominent part in the bradycardiac crises. In bradycardia occasioned by impairment of the intracardiac conductivity digitalis is distinctly contra-indicated.

A pulse slightly retarded by increase of the contractile irritation at the venous openings, of which only every second or third is transmitted to the ventricles, may possibly be influenced under normal conditions of conductivity by digitalis in combination with atropin. Bradycardia caused by intestinal auto-intoxication requires intestinal antiseptics, as calomel. If the cause of the slow pulse is found to be an inhibition of conductivity, inhalation of oxygen would be justified in view of the experimental experience gained with asphyctic frog hearts.

Pulse retardation developing in an acute dilatation of the heart may sometimes require venesection in threatening edema of the lungs. If bradycardia is accompanied by dyspnoea and diminished diuresis a morphine injection may produce sudden death. In these cases where, on account of the pronounced dyspnoea, morphine is indispensable, a simultaneous injection of caffein or atropin, as the case may be, is indicated. General therapeutic indications cannot be given, as each concrete case requires individual analysis as it arises.

PART II

TACHYCARDIA

II

TACHYCARDIA

Conditions which Induce Tachycardia.—Considerable increase in the number of heart-beats, together with a correspondingly increased abnormal pulse frequency, is designated as tachycardia in clinical medicine.

The number of heart-beats in the adult is 65-75-80 per minute under normal circumstances. In the first year of life the pulse beats 120 to 130 times per minute; in the very old there is a slowing of the cardiac action without pathological changes in the heart.

Under the influence of digestion and bodily exertion, of mental overwork, and of high temperature (as in tropical climates), there is accelerated cardiac action even under normal conditions. The number of heart-beats increases during inspiration and decreases during expiration. The well-known experiment of Hering, in which inflation of the lungs of a dog caused lowering of the blood pressure and acceleration of the cardiac action, has been explained by excitation of the sensory nerve fibers of the lungs, which by reflex action reduce the tonus of the inhibitory fibers. Analogous to this experiment is Valsalva's procedure applied in otology, which consists of forced expiration with closed mouth and nostrils, resulting in a simultaneous lowering of the blood pressure and acceleration of the pulse.

It has long been known that changes in the rhythm of respiration directly influence the frequency of the pulse. Frequent, superficial respiration accelerates the cardiac action, while less frequent respiration, interrupted by deep inspirations, effects a retardation in the heart-beats. In the initial stage, however, of laryngotracheal stenosis the pulse frequency is increased during the protracted inspiration.

Voluntary acceleration of the cardiac action with unchanged rhythm of respiration has also been repeatedly observed. A colleague whom I examined was able by mere exertion of will power to accelerate his heart action from 70 to 110 during perfectly normal respiration while the experiment lasted. There was a moderate accentuation of the second pulmonic sound, while the tension of the accelerated pulse was not weaker, but rather stronger, than before the experiment. The borders of the heart remained normal during the experiment. The acceleration of the pulse did not take place all at once, both the increase and decrease being gradual. This would indicate that in this case the cardiac center of inhibition, situated in the oblongata, was not paralyzed, but rather that the cardiac center of acceleration was excited: a behavior which corresponds perfectly with the acceleration of the heart-beats through irritation of the accelerans.

Tachycardia occurring during pregnancy, after parturition and during lactation, should be regarded as physiological, and is therefore not included in this presentation of the subject.

TACHYCARDIA IN FEBRILE AFFECTIONS.—At the bedside tachycardia is observed as a symptom in a great many different diseases. Acceleration of pulse and fever are so intimately connected with each other that Liebermeister has expressed their reciprocity in this formula: Pulse=80 + 8 (Temp. — 37). If we consider that tachycardia in febrile affections is not only the consequence of increased temperature, but is dependent on the nature of the infection or its toxins; that furthermore the quantity of the poison present in the circulation, the individual reaction of the patient, and his circulatory organs are to be taken into consideration, it is easily perceived that Liebermeister's formula cannot apply in all cases.

In many febrile infectious diseases there are striking divergences between the temperature and pulse, the latter either not rising in proportion to the temperature, or being disproportionately quicker. These deviations between pulse and fever, which French authors call "fièvres dissociées," depend partly on the nature of the infectious agent and its effect upon the heart and vessels, and in part on the injury to the circulatory apparatus which it produces, and lastly on the condition of the heart previous to the infection. All these factors, including the individual reaction, should therefore be taken into consideration in passing a prognostic judgment of tachycardia accompanying febrile infectious diseases.

TACHYCARDIA IN TYPHOID FEVER.—In uncomplicated cases of typhoid fever in strong adults, particularly males, the curves of pulse and temperature are generally parallel, but the number of pulse beats is but slightly increased in comparison with the rise in temperature, even in the gravest cases, accompanied by diarrhea, meteorism, and impaired sensorium. Temperatures of 39°-40°, and even higher, are seen in typhoid fever simultaneously with a pulse rate of 70-90. This comparative slowness of the pulse characteristic for normal typhoid fever, together with its likewise characteristic dicrotism, is of especial diagnostic importance in differentiating this affection from gastric fever, influenza, tuberculosis, acute endocarditis, and septic infections, in which, as a rule, the pulse rate increases disproportionately more than does the temperature. For this reason a continuously and considerably accelerated pulse both at the beginning and in the course of typhoid is a prognostically unfavorable sign, especially if the blood pressure at the same time decreases. A brief acceleration of the pulse, however, especially in neurasthenic patients in poor condition, in women and children, after bodily exertion, or psychic excitation, is usually without significance during the course, and especially in the final stage and in convalescence, even if the rate rises to 120 or 130. An acceleration of the pulse to 130 to 140 in strong men is nearly always an ominous symptom.

Continuous tachycardia occurs in the adynamic forms of typhoid fever, while sudden acceleration of the pulse in conditions of collapse, as after intestinal hemorrhages or in complications with peritonitis, are well-known manifestations, and justify an absolutely unfavorable outcome, especially with rapidly declining temperature.

The relative bradycardia which is present in the normal course of typhoid is evidently due to the effect of the typhoid toxins upon the nervous system, and perhaps on the heart itself. It must be assumed that the typhoid poison acts analogously to digitalis or the biliary acids.

In grave cases of typhoid, in which the tension and fullness of the pulse lessens at an early period, it may be imagined that the typhus poison in large quantities or in strong concentration exerts a paralyzing influence upon the heart and vasomotors. The mere lowering of the blood pressure caused by paralysis of the vasomotors may alone be sufficient to increase the frequency of heart-beats by diminishing the peripheral resistance. Tachycardia in typhoid may therefore result from primary lesions of the heart, as well as, secondarily, from vasomotor paralysis, with heart intact.

These considerations may lead to important therapeutic indications in a given case, as hydropathic procedures and influencing the vasomotors by vascular poisons (ergotin, strychnine, adrenal preparations). In mixed affections with bacillus coli, and especially streptococci, the effect of their toxins in accelerating the pulse must naturally be taken into consideration.

Tachycardia in Typhus Fever and Relapsing Fever.—In contradistinction to the above described behavior of the pulse in typhoid fever the pulse frequency in typhus fever both at the beginning and during the course of the disease is considerably increased; even in strong men the pulse rises above 100 in the evening, and seldom sinks below 100 in the morning. Nor is a dicrotic pulse a frequent manifestation.

This symptom which characterizes typhus fever is in grave cases due principally to changes in the myocardium. The complete dissimilitude of the poison of typhus fever possibly plays a special rôle, if further investigations should demonstrate that typhus fever, in accordance with Gotschlich's findings, is caused by a parasitic protozoon (apiosoma) in the red blood corpuscles.

The tendency to tachycardia appears even more marked in the paroxysms of relapsing fever than in typhus fever. A pulse rate of 100 to 140 per minute is the rule, and even 140 to 160 beats by high temperatures are not infrequent. Toward the end of the paroxysms at the time of the crisis pronounced tachycardia with thready and irregular pulse and collapse-temperature has been observed in cases which went on to recovery.

Tachycardia in Influenza.—Influenza is also an affection in which acceleration of the pulse is practically the rule, is often disproportionate to the temperature and sometimes even occurs in afebrile cases. Even in perfectly healthy persons, in whom neither fatty heart, valvular insufficiency, nor arterio-sclerosis is demonstrable, the pulse sometimes shows striking frequency and smallness. Even after la grippe has run its course, tachycardia and softness of the pulse, with palpitation, oppression, irregular cardiac action and dyspnoea, has been observed. Both the tachycardia and paroxysms of cardiac weakness are undoubtedly dependent on changes in the myocardium. In some cases where pulse acceleration occurs without congestion of the lungs, liver, or kidney, at times even in an apparently light, uncomplicated attack of influenza, and remains present after the recovery from the illness, it is attributable to changes in the cardiac nerve apparatus. The pulse acceleration in these forms may be so pronounced that some, notably French, authors (Peter, Huchard) have distinctly assigned its cause to a direct vagus paralysis. The possible presence of bradycardia, together with the possibility of the latter changing into tachycardia, has already been mentioned in the preceding part on bradycardia. The latter phenomenon, together with the labile frequency of the pulse is possibly due merely to an irritable weakness of the vagus. In other cases it may be due to anatomical changes in the myocardium itself. Increased attention should be paid to both bradycardia, and especially tachycardia, for not infrequently they are the forerunners of grave attacks of impending cardiac weakness and death from acute heart disease.

Tachycardia in Diphtheria.—The insidious character of these at times ominous disturbances of cardiac innervation is still more apparent in another acute infectious disease, namely, diphtheria. The diphtheritic toxin is a poison which not only directly attacks the nuclei of the myocardiac cells and dissolves the muscle fibers (Eppinger), but also injures the vasomotors. This sufficiently explains the reduced cardiac function on the one hand and the poor arterial tension on the other, even in cases of only moderate gravity. This also makes clear various anomalies of cardiac function and innervation in a satisfactory manner. Tachycardia, arhythmia, dullness of the heart sounds, gallop-rhythm, are manifestations of the damage done to the circulatory apparatus by the toxins of pure diphtheria. They are not infrequently prodromata of death from acute cardiac disease while the local diphtheritic changes are still present, in contradistinction to the cardiac attacks—designated as secondary cardiac paralysis—after the local process has subsided in the third or fourth week of the illness. Suddenly, and while enjoying apparent euphoria, patients collapse, bradycardia and tachycardia occur, then arhythmia, dull heart sounds, vomiting, cold perspiration, pallor of the face and other signs of impending death from syncope. These catastrophies, known as late paralyses, may be caused by degeneration of the myocardium as well as by neuritic changes in the vagus.

Tachycardia in Pneumonia.—In the croupous pneumonia of adults the pulse frequence attains 100 beats per minute and more. The proportion between respiration and pulse frequency, which in the healthy is 2 : 9, is disturbed in croupous pneumonia so that the frequency of respiration rises relatively higher than that of the pulse. Cases in which the frequency of pulse is higher than the corresponding state of the fever, and still more cases where with relatively low temperature the pulse frequence corresponds to that of the respiration, give rise to prognostic anxiety. As a rule, however, the frequency of both pulse and respiration in croupous pneumonia does not attain to that striking height characteristic of acute miliary tuberculosis of the lungs. Patients whose hearts were already degenerated or otherwise affected before the outbreak of the disease are exceptions to this rule. In pneumonia of this kind there is tachycardia with small, soft or large, easily compressible and frequently dicrotic pulse, weakness of heart sounds, especially of the second aortic sound, feeble or absent cardiac impulse. A similar picture is presented by the so-called asthenic pneumonia, in which tachycardia, with not excessive temperature, with swelling of the spleen and liver, icterus, meteorism, nephritis, and prostration justify the assumption of an intense intoxication.

In complications of pneumonia with pericarditis, also in old and decrepit individuals, sometimes, too, in the initial stage of pneumonia which develops into pulmonary gangrene; again in the pneumonia of diabetes, of poisoning with carbon monoxyde, of tuberculous patients, and in bronchopneumonia, especially after influenza, the plague, and trichinosis, there is abnormally high pulse frequency, together with great dyspnoea and cyanosis. Such inflammation of the lungs may present difficulties in differential diagnosis from miliary tuberculosis, in which a remarkable increase in frequency of respiration and pulse is the characteristic picture. Tachycardia with fatal collapse may occur during the convalescence of pneumonia in patients who leave the bed too early.

From these considerations it is apparent that the acceleration of cardiac action in pneumonia is the result of various factors. It is dependent on the degree of injury to the heart, on the extent of the pneumonic process, on the individual affected, on possible complications (pericarditis, endocarditis, pleuritis), and not less also on the intensity of the intoxication with the poisonous products of the causative factor; even the nature of the latter may not be without influence upon this phenomenon. The metabolic products of the streptococcus in particular seem to produce the most pronounced tachycardias. In septic affections due to streptococcic infection there is a remarkable acceleration of the pulse even with an afebrile course. This is particularly noticeable in streptococcic peritonitis in the puerperium, as compared with peritonitis caused by staphylococci and bacteria coli. The same holds good for tachycardia in erysipelas in different locations, in which, however, the involvement of the heart or the localization at the valves (myocarditis, endocarditis) plays a prominent part.

TACHYCARDIA IN MALARIA, SCARLET FEVER, VARIOLA.—In the stage of apyrexia of malaria when the pulse frequency after a rise does not return to normal, tachycardia may give rise to the suspicion that in addition to malaria there is a complicating septic infection. I recall such a case, a tertian malaria in the puerperium, complicated by a streptococcic infection. It was the persistent tachycardia during the apyrexia, together with the leucocytosis, which led to the correct diagnosis of the complication. There developed thrombosis of the saphena and a parametritic abscess which voided through the vagina pus containing streptococci demonstrable in pure culture. Streptococci also possibly play a rôle in the tachycardia of scarlet fever which gives to that affection a certain characteristic as distinguished from measles. In the very beginning of scarlet fever there is a pulse frequency of 160 to 180, in babies even of 200 beats per minute, and this, too, in cases which go on to recovery. Unusual pulse frequency, however, when small and irregular, associated with coldness of the extremities and of the tip of the nose, is even in scarlet fever a prognostically ominous symptom.

These examples indicate the important prognostic significance of pulse acceleration as an early symptom in incipient disease. If, after a chill, there is an abnormally high temperature and an unusual acceleration of the pulse, and if this tachycardia is the forerunner of scarlet fever, we know that the disease will be severe, though not necessarily fatal. If, however, pneumonia follows, it means in the majority of cases a very grave disease, which in the adult is nearly always fatal.

In moderately severe cases of variola there is, as a rule, no such pronounced tachycardia as in scarlet fever. The pulse frequence corresponds to the rise in temperature, and in robust individuals it is full and tense. Intense tachycardia with small, compressible pulse, delirium, coma, and abnormally high temperature occurs in those infectious diseases which are fatal even in the initial stage. In the grave forms of variola hemorrhagica which commence with little or no fever there is an early and abnormal acceleration of the pulse, which is small, soft, irregular, or intermittent. Similarly, tachycardia with small, irregular pulse occurs in the stage of suppuration as an expression of the cardiac weakness, with stupor, stertorous breathing, both in hyperpyretic cases and those accompanied by collapse-temperatures, especially in variola pustulosa hemorrhagica.

TACHYCARDIA IN ACUTE ENDOCARDITIS.—In acute endocarditis the pulse frequency is subject to great variations, which are due partly to the rise in temperature, in part, however, to the obstructed breathing from pulmonary complications. It is regular and irregular, intermittent, dicrotic, exceedingly frequent, and also small. In the majority of cases, however, there is a tendency to tachycardia, especially after psychic disturbances and still more after bodily movements. Traube has emphasized the prognostic importance of this acceleration of the pulse. The persistency of tachycardia in the course of an acute endocarditis points to the fact, as has already been explained in the part on bradycardia, that the endocarditic process has not yet come to an end, so that during convalescence a premature getting-up may be fatal to the patient. In the subfebrile and afebrile types of endocarditis there is also frequency of pulse, especially in the septic forms, accompanied by pronounced anemia. This is also true of the apparently afebrile cases of pleuritis, especially the ambulatory form, in which patients do not experience any special distress in the chest, and where examination reveals a pleural exudate. In dry pleurisy, and still more in pericarditis, an unusually rapid pulse is sometimes met with in ambulatory patients, and therefore tachycardia in this class of patients demands a thorough examination of the thoracic organs. A marked acceleration of the pulse is frequently found in pneumothorax. A striking example of this form of tachycardia was presented last year in a patient with left-sided tuberculous pyopneumothorax who was free from fever, and in whom the test puncture showed sterile pus.

TACHYCARDIA IN TUBERCULOSIS.—In subfebrile or afebrile pulmonary tuberculosis tachycardia is not infrequent, and under all circumstances is of unfavorable prognostic significance. It occurs in advanced tuberculization, and may be caused by atrophy of the heart, and in some cases by compression of the vagus by degenerated tuberculous glands. Tachycardia is also frequent in the apyretic wasting of diabetes, in that of pericardial adhesions, and in terminal exhaustion of patients with heart disease, as affections of the pulmonary valves and aneurisms.

Tachycardia in tuberculosis may also be associated with impaired respiration from extensive infiltrations of the lungs, but this is not always true, as the following case shows: A hackman, 37 years of age, had been under observation for fourteen days, and only once showed an afternoon temperature of 37.8°; at other times always below 37.6°. The pulse was small, soft, 120; respiration, 24, and without dyspnœa. The autopsy revealed a small cavity with soft, caseous walls and slightly diffused apex tuberculosis. The pus from the cavity contained exclusively tubercle bacilli—no diplococci or streptococci. This case, which must be looked upon as a purely tuberculous caseous infiltration, is not uninteresting in considering the question of tachycardia in tuberculosis, for the reason that Bouchard has isolated a body from tuberculin which possesses distinct vaso-dilatory properties. Tachycardia may therefore be the direct sequel of the diminished blood pressure caused by vaso-dilatation, as is positively observed in many cases of tuberculosis. It appears, therefore, that the toxins of the tubercle bacillus are able, by reinfections, to accelerate the cardiac action in a purely toxic way without complications on the part of the heart or its nerves.

TACHYCARDIA IN MIXED INFECTIONS; COLON BACILLUS.—In mixed infections, such as occur in cavernous phthisis, the nature of the other causative factors is naturally also of importance for the genesis of tachycardia, especially streptococci, whose pulse-accelerating properties have already been mentioned; mixed infections with the coli group do not seem to have any material influence upon the cardiac action.

In colibacillosis, as in a febrile cystopyelitis affecting a patient in our clinic, in which the agglutination of the culture grown from the urine justified the diagnosis of an infection with bacillus coli, the pulse was no more accelerated than the fever justified. The same is true in those rare case of peritonitis caused by the bacterium coli, in which an acceleration of the pulse such as is characteristic in streptococcic peritonitis does not take place. From this it seems probable that the toxins of the bacterium coli behave differently in resorption by the peritoneum than do the streptococcic toxins. In those forms of peritonitis caused by staphylococci—usually non-aggressive to the peritoneum—acceleration of the pulse plays no prominent rôle, even if the process becomes generalized, while in streptococcic peritonitis a small, uneven, exceedingly frequent pulse of 150 or 180 and upward is a characteristic sign of sepsis. In these cases normal temperature and painlessness of the abdomen may erroneously lead to an apparently favorable prognosis, especially in patients to whom previously opium had been administered. Occasional bilious vomiting, striking divergence between pulse frequence and temperature, and coldness of the extremities justify the worst prognosis of a septic intoxication. In suppurative forms of peritonitis which lead to abscess formation in Douglas' space, extreme divergences between pulse rate and temperature are not observed as in diffuse septic peritonitis. This relation of temperature to pulse frequence has a peculiar significance for the diagnosis and prognosis of acute appendicitis. In the differential diagnosis of limited and disseminated peritonitis it plays an important rôle. In general, cases of appendicitis in which the pulse rate exceeds 120 a minute justify in the large majority of cases absolutely unfavorable prognoses.

Tachycardia in Cholera.—Cholera Asiatica and cholera nostras belong to the acute infectious diseases which begin and continue without fever, and in which the small and frequent pulse, with impaired circulation and collapse, are symptoms. This applies not only to the cold stage of cholera, but also to cholera typhoid, in which there is fever and typhoid prostration, together with dicrotic and—in case of very high fever—exceedingly frequent pulse, in contradistinction to typhoid.

TACHYCARDIA IN NON-FEBRILE AFFECTIONS; THE DRUG INTOXICATIONS.—While febrile affections have been mainly mentioned in which tachycardia occurred consequent upon increased temperature and the toxins of infectious diseases, there is also an acceleration of pulse without rise in temperature in many acute, sub-acute, and chronic affections, among which the intoxications form a special group. In the great majority of severe poisonings, as in arsenic and phosphorus poisoning, there is an acceleration of pulse in the terminal stage caused by a secondary injury to the circulatory apparatus.

Our next subject of consideration will be poisons which, when continuously administered in small doses, cause disturbances of circulation and thus lead to more or less permanent tachycardia; or sub-acute intoxications, in which even relatively small doses attack especially the heart, or rather the cardiac nerve center. The chief representative of the latter group is atropin, which, as is well known, causes acceleration of the pulse both in animal experiments and in man, owing to its paralyzing effect upon the vagus terminals. A patient was brought to Duchek's clinic who had been taken ill on an excursion into the country, and presented the picture of alcoholic intoxication. As he acted like a maniac and frustrated every attempt at examination, he was transferred to the psychopathic ward, with the diagnosis of delirium potatorum. When the patient began to quiet down, there was noted enormous dilatation of the pupils, accelerated pulse and intense hyperemia of the skin. He had eaten wild cherries, which had occasioned his condition.

Transitory acceleration of the pulse with pronounced dicrotism and reduced blood pressure is observed after the medicinal use of amyl nitrit and nitroglycerin; a similar effect is brought about by potassium iodid and thyreo-iodin. Acute poisoning with digitalis or the cumulative effect of the same at first produces retardation of the pulse, which may be changed to tachycardia with arhythmia and weakness of the heart. Similarly acute opium and morphine intoxication may frequently induce an accelerated pulse up to 180, in addition to dyspnœa, stertorous breathing, coma, and markedly contracted pupils. In the careless injection of morphine into a vein, the pulse may become exceedingly frequent, and the same effect may follow the too rapid withdrawal of morphine, which may occasion tachycardia and general collapse. A similar effect is produced by cocain intoxication, especially in neurasthenic persons, also after the overindulgence in coffee and tea, which causes acceleration of the pulse, with small and at times intermittent beats, insomnia, tremor, vertigo, and sensation of fright. In chronic alcoholism tachycardia may be produced by the toxic action upon the vessels as well as by secondary lesions of the heart, or by neuritis of the vagus, which latter condition is possibly a part manifestation of alcoholic polyneuritis, as described by Dejerine. I have just recently seen such a case with dilatation of the heart, dull cardiac sounds, very rapid pulse rate, swelling of the liver, edema of the lower extremities, and marked pallor. He was a youthful drunkard who entirely recovered in an institution upon the withdrawal of alcohol.

The tachycardia of tobacco smokers, which is especially evident after bodily exertion, and is accompanied by an intermittent pulse, has been discussed in the section on bradycardia.

Of the forms of tachycardia which occur in autointoxications, that one should be singled out which persists until death and is observed in diabetic coma.

Tachycardia in Affections of the Heart.—Various disorders of the circulation may be accompanied by tachycardia. It is oftentimes the only symptom of an overstrained heart as a concomitant manifestation of its acute dilatation. G. Sée described a type of tachycardia in hypertrophy of the heart in the period of development (hypertrophie de croissance). Permanent acceleration of the pulse, even without arhythmia or intermissions, has been observed in acute and chronic myocarditis. I had occasion to see such a case. He was a person of advanced age, in whom considerable acceleration of the pulse without arhythmia was the foremost factor in the clinical picture, in addition to signs of cardiac dilatation, cyanosis, and signs of general congestion. In arterio-sclerosis of the heart tachycardia may likewise be permanent and attain an extreme intensity in individual cases, especially in sclerosis of the coronary arteries. In angina pectoris pulse acceleration is not infrequent as the paroxysm subsides; in valvular insufficiency it is frequently observed. In mitral affections, like mitral stenosis, and in myomalacic inefficiency of the heart, tachycardia is found most frequently in conjunction with arhythmia, which latter, however, is much less frequent in insufficiency of the aortic valves.

In productive endocarditis of the aortic valves tachycardia may appear as a terminal manifestation. I observed such a case, in which the formation of vegetations led to a sudden occlusion of the aortic orifice. Similarly, acute aortitis may be accompanied by spasmodic acceleration of the pulse up to 120-140. To this class of cardio-vascular tachycardia Dehio's case evidently belongs. He describes the acceleration of the pulse up to 180 in a patient suffering from hepatic cirrhosis after puncture of a large ascites. The weak heart, together with the sudden lowering of the intra-abdominal pressure, may have been the causative factor.

TACHYCARDIA IN AFFECTIONS OF THE VAGUS.—Affections of the vagus and the vagus center in the oblongata are among the principal causes of symptomatic tachycardia. There is either a direct anatomical lesion of the affected nerve or a compression which first causes irritation and later on paralysis of the nerve. The high frequency of the pulse in the terminal stage of meningitis is explained by the paralysis of the vagus center. Hydrocephalus, caries of the atlas, constricting affections of the fourth ventricle, tumors of the medulla and of the cerebellum, also tumors of the cerebrum, may have a distant effect upon the vagus center. It is just in these infections that bradycardia and tachycardia alternate; indeed, very pronounced bradycardia may suddenly turn into tachycardia of like intensity. In bulbar paralysis, Duchenne has already described extreme acceleration of the heart with irregularity of the pulse. In the same way paralysis of the vagus center explains the abnormal frequency of the pulse by softening of the oblongata due to thrombosis and embolism. Senator observed a case of bulbar paralysis of the vago-accessorius in thrombosis of the vertebral artery, accompanied by tachycardia, dysphagia, snuffling speech, sensation of hunger and of oppression, objective dyspnœa being absent. Affections of the nervous system, as Landry's paralysis, acute myelitis, anterior poliencephalitis, amyotrophic lateral sclerosis, disseminated sclerosis, and hemorrhages of the fourth ventricle may cause tachycardia, should they attack the vagus nucleus. Charcot has already pointed out in tabes the increased pulse rate which may appear as an early and a late symptom or simultaneously with the "crises gastriques." That these forms of tachycardia may be explained by neuritic affections of the vagus is proved by a case, in point, of Oppenheim. In syringomyelia also there is accelerated pulse, in addition to other bulbar manifestations.

Compression and anatomical lesions of the vagus trunk on leaving the medulla are often causes of tachycardia, as pressure by tumors, by syphilitic growths of the meninges, osseous tumors, caries of the base of the skull, aneurisms, hematoma of the dura in fractures of the base of the skull, or cerebellar tumors. I found an abnormally high pulse rate, with vomiting and right-sided paralysis of the hypoglossus, after a cancerous metastasis in the foramen jugulare vagi. Tachycardia has been observed also in thrombosis of the vena jugularis after suppurative inflammations of the middle ear. Pressure of a thrombus situated in the fossa jugularis upon the vagus, glosso-pharyngeus, accessorius and hypo-glossus may produce manifestations of irritation and paralysis in the area of these nerves, as aphonia, dysphagia, and cough.

The vagus in its course may be compressed or injured by abscesses of the neck, phlegmonous processes, glandular and other tumors, cicatrices, aneurisms of the carotid and subclavian, injuries sustained from shooting, stabbing or blows, in operations on the neck (extirpation of struma), and tachycardia results. In the mediastinum also the vagus may be similarly affected by glands, tumors, aneurisms of the aorta and innominate artery. In tubercular subjects a pulse acceleration occurring with spasmodic, pertussis-like coughing, directs suspicion to tuberculosis of the bronchial glands which compress the vagus. Several such cases have been mentioned in the literature, and I had occasion to see one which was complicated, in addition, with uncontrollable vomiting. Also in infectious diseases, with swelling of the peribronchial and mediastinal glands, in measles, pneumotyphoid, pertussis, and diphtheria, this cause of pulse acceleration should be kept in mind.

Pertussis, complicated with swelling of the tracheobronchial glands, but without pulmonary complications, has shown a pulse rate of 130 to 140, although the temperature was comparatively low. When complicated with broncho-pneumonia in children, the number of pulse beats may reach 160, 180, and even 200, and that without excessive fever. Tachycardia in pertussis should therefore direct attention to a broncho-pneumonia, cardiac weakness, or glandular swelling in the mediastinum.

In carcinoma and lymphosarcoma of the mediastinum, pressure on the trachea, vagus, recurrent and phrenic nerves may impart great diversity to the clinical picture: spasms of the glottis, vomiting, tachycardia, singultus, asthmatic attacks, dysphagia, as in the well-known manifestations of aneurisms of the aorta. All these compressions of the vagus may not only be mechanical, but also due to an inflammation of the nerve sheath. Pleuro-pericardial inflammations and glandular swellings from adenitis and periadenitis may finally develop a vagus neuritis. Other affections of the vagus which may bring about tachycardia are the various forms of vagus neuritis, which not only occur in the course of infectious diseases, but also follow in the wake of intoxications. All these forms belong to the group of polyneuritis, and it may suffice to single out one case, observed a few years ago. The patient was a painter's apprentice, whose most pronounced symptom, aside from cerebral and dyspeptic manifestations, was vomiting, with hematemesis. The initial stage, with predominating cerebral manifestations, as cephalalgia, pain in the nape of the neck, and slight delirium, suggested tubercular meningitis. In the further course, tachycardia became especially noticeable, and had the peculiarity that it was not preceded by bradycardia, as is usual in exudative processes of the meninges. The hematemesis which occurred at a later stage differed from that usual in gastric ulcers principally by the fact that there was no free hydrochloric acid in the vomited matter, so that a latent, preexistent ulcus ventriculi was not the probable cause of the gastric hemorrhage. There developed in addition a lobular pneumonia. In critically reviewing the case, especially in view of the patient's occupation as painter, the following diagnosis was reached: Neuritis vagi saturnina. The unusual tachycardia, as well as the later broncho-pneumonia and the gastric hemorrhages, were thus interpreted as vagus phenomena. The autopsy disclosed gray degeneration of the left vagus and a circumscribed softening of the gastric wall; the latter was also considered the sequence of a vagus lesion by the pathologists. All these forms of tachycardia caused by compression or anatomical lesions of the vagus trunk, are characterized by their persistence as opposed to the symptomatic tachycardias of different origin, especially the reflex forms, in which the pulse acceleration is either transitory or irregularly variable, as is the case in the tachycardia of cardiac disease. This statement, however, does not apply to all cases of vagus tachycardia, because there are also forms caused by vagus compressions which also alternate with bradycardia. In this connection a case is interesting which was treated about eight years ago in our clinic and published by Ortner. There was bilateral tubercular infiltration of the lungs, left adhesive pleuritis and swelling of the cervical lymphatic glands. Patient had considerable tachycardia, 140 to 160 beats per minute, also meteorism, which disappeared as tachycardia subsided. The tachycardia corresponded to the distention of the abdomen.

The result of the autopsy was: Bilateral tuberculosis of the lungs, left pleuritic adhesions, complete incarceration of the left vagus in a tubercular mediastinal lymph gland. These observations indicate that even in pronounced anatomical changes of the vagus, tachycardia is not always permanent, but may be intermittent. This, however, appears possible only in unilateral affections of the vagus.

Compression and degeneration of both vagi manifest by no means greater pulse frequency than do unilateral lesions. Tachycardia therefore seems to depend on the character of the lesion, especially of the cardiac fibers. It can easily be understood that in most cases of lesions of the trunk of the vagus there are also manifestations on the part of the lungs, larynx, and gastro-intestinal tract. It seems always remarkable that on compressing the vagus at the same place, for instance, in the neck, the symptoms differ—one time, the gastric, another time, the cardiac or pulmonary are the most prominent. Again, cases in which the vagus was cut during operative procedure have been known to show not the slightest change in the pulse rate. Possibly these variations in clinical symptoms depend on unequal distribution of the cardiac, pulmonary and intestinal fibers, to both trunks of the vagus. An important characteristic of the tachycardia produced by either unilateral or bilateral vagus lesions consists in the fact that the heart action always keeps within moderate limits, on the average between 120 and 150, and that according to all previous observations 160 has never been exceeded. The blood pressure is not lowered and cardiac action is perfectly regular. Even in poisoning with atropin, which paralyzes the intracardiac vagus ends, there is, as a rule, only moderate acceleration. In one case of atropin poisoning, in which the pulse rate was 180, the beats were absolutely regular. The blood pressure in atropin poisoning is also raised.

TACHYCARDIA IN CACHEXIAS.—Symptomatic tachycardia occurs also in chronic cachexias. In the cachexia of tuberculosis permanent acceleration of the pulse may, as we have learned, have different causes. In many cases it is the progressive atrophy and degeneration of the heart, which may be the cause not only of the tachycardia, but also of the pre-mortal embryocardia and death.

Tachycardia may be an early symptom of latent pulmonary tuberculosis. If it is accompanied by emaciation, which cannot be explained by diarrhea, loss of fluids, diabetes, cachexia or otherwise, the suspicion of incipient pulmonary tuberculosis, even when there is no fever, should be aroused. These forms of tuberculosis justify a grave prognosis.

Similarly, tachycardia occurs in the final stage of other cachexias, as carcinoma, amyloidosis, et cetera; hemorrhages, if any occur, may be of importance. In chronic and pernicious anemia, leukemia, or pseudo-leukemia, tachycardia, in our experience, does not occur particularly frequently, in spite of the grave degeneration of the myocardium. An abnormally rapid pulse in leukemia and pseudo-leukemia may, without doubt, be due to compression of the vagus by enlarged lymph glands in the mediastinum. In chlorosis there is both a frequent, soft pulse and palpitation, especially after bodily exertions and psychic excitement. These forms of tachycardia generally disappear with rest and during sleep, and thus differ from those observed in Basedow's disease.

TACHYCARDIA IN BASEDOW'S DISEASE.—Tachycardia, as is well known, is one of the early and cardinal symptoms in exophthalmic goiter, although there are a very few cases where this disease begins with struma or exophthalmus, and the accelerated pulse develops only in its later course. The permanently accelerated pulse in Basedow's disease remains unchanged with rest and does not disappear during sleep, in contradistinction to the tachycardia of neurasthenia and hysteria. In excitement and physical efforts the acceleration may even increase in this disease.

Spasmodic increase of the pulse rate to 160, 180, and even 200, is frequently observed. F. Müller reports a case of Basedow's disease which terminated fatally with very acute manifestations and a pulse rate of 186. Here there was considerable degeneration of both vagus trunks in the cervical region.

Digitalis does not influence the pulse rate in this disease. In a case of Gerhardt, in which digitalis remained without effect, there was later, when patient became icteric, a retardation of the formerly accelerated pulse, evidently under the influence of the biliary acids upon the heart. In some cases of Basedow's disease the pulse rate is higher in the morning than in the evening. Cases are by no means rare in which the enlarged thyroid is very slight and the tachycardia especially marked. The pulse rate does not always keep step with the exophthalmus or with the goiter. Both the thyroid and the exophthalmus may be prominent, yet the pulse is at times between 90 and 100, while with small thyroid and slight exophthalmus it has been observed as high as 150. The tachycardia in Basedow's disease may become very pronounced if in its course an infectious disease supervenes, as influenza, strumitis, or intestinal auto-intoxication. There may, however, be febrile rises of temperature in Basedow's disease, and then the tachycardia may present great difficulty in diagnosis, especially in cases where the disease is not pronounced, but is of the type of the so-called "formes frustes." Especially if there is great emaciation, diarrhea which suddenly appears and as quickly disappears, or complications with Addison's disease, the correct clinical diagnosis and the prognostic interpretation of tachycardia may be very difficult. According to Charcot, tachycardia which progressively declines for several weeks is a favorable sign for ultimate recovery, in spite of the persistence of the other Basedow symptoms.

Tachycardia in Neurasthenia, Hysteria, Traumatic and Other Neuroses.—Tachycardia in so-called neurasthenic cardiac weakness has been observed up to 150. It commences and disappears gradually, and in contradistinction to paroxysmal tachycardia shows other peculiarities, such as striking acceleration of the pulse, which becomes smaller, and even disappears during inspiration; also arhythmia caused by extra systoles. A short time ago I observed this behavior of the pulse in a young neurasthenic theologian. At the same time there were asthmatic paroxysms, with expectoration of sputum containing plentiful Curschmann's spirals, but neither abundant eosinophile cells nor Charcot's crytals were demonstrable. It should be mentioned that in this case both the tachycardia and arhythmia were arrested by injection of atropin. A pronounced irregular heart, as is sometimes observed in a neurasthenic cardiac neurosis, was not present. A case of tachycardia up to 120, which had existed for some time, occurred in a hysterical and somewhat adipose lady, who was under the delusion that she was suffering from Basedow's disease, which idea had been strengthened by the reading of some medical books. As all the other symptoms of Basedow's disease were absent, a diagnosis of hysterical tachycardia was made. Cases of this kind, which occur rather frequently in medical practice, including also traumatic neuroses, where a considerably accelerated pulse is the rule, require prolonged observation to establish the true condition.

In traumatic neurosis a pulse of 120 and more is not a rare occurrence, especially after slight bodily exertion or psychic excitement. Oppenheim and Lehr reported several such cases, where there was considerable increase in the pulse rate when the patients told of the accident that had befallen them. The lability of the pulse was also demonstrated by the use of Mannkopf's method, which consists in pressure on painful points, which may increase the pulse rate by thirty beats a minute.

To explain the tachycardia in the last mentioned neurosis, those cases are significant in which tumors of the motor zone and cortical epilepsy show considerable acceleration of the pulse, and also the experiment of Franck, who succeeded in producing tachycardia by cortical irritation. It would seem possible that psychic influence upon the center of the cardiac movement in the medulla oblongata, practically in an auto-suggestive way, might both create and dispel disturbances of cardiac action and perhaps more or less permanent pulse acceleration. This is also demonstrated in those rare cases where individuals are capable of spontaneously increasing their heart-beat. The positive effect of suggestive treatment may perhaps throw light on this subject, especially if a rudimentary form of Basedow's disease can be excluded. Epilepsy is now and then characterized by transitory tachycardia both in the beginning and in the later part of the paroxysms. Hoffmann has seen tachycardia acting as an equivalent to migraine, with which it alternated. In chorea acceleration of pulse up to 120 or 130 has been observed at times even by slight movements. Finally, those forms should be mentioned which Friedrich Kraus describes as a consequence of exaggerated function of a hyperplastic thyroid under the name of "goiter heart," which he separates from the symptom complex of Basedow's disease. Tachycardia occurring during the exaltation of mania may be caused by various factors, which, however, do not play a material part in the clinical picture. Numerous are those forms of tachycardia which have been described as reflex and emanate especially from the abdominal organs, in dyspepsia, hyperchlorhydria, achlorhydria, intestinal parasites, and intoxications after chilling the skin, notably those which arise from the sexual organs. Permanent tachycardia, accompanied by sensations of fright, temper, heat, vertigo, and profuse perspiration, is a frequent manifestation at the climacteric, which sometimes at the commencement presents a pathological picture reminding one of Basedow's disease. The pulse rate may rise to 120 to 150 and at times more; the pulse, however, remains regular, strong, and well filled. Castration of mature women may also be followed by attacks of tachycardia. Typical Basedow's disease has appeared after bilateral ovariotomy in a young woman. Thomson describes several cases of persistent tachycardia up to 160, associated with nausea, vertigo, anorexia, etc., and believes that the eating of raw meat may easily cause these manifestations. Treatment consisted in the administration of milk, vegetable diet, and an occasional dose of calomel. In diabetic coma progressive tachycardia up to the end is a well-known clinical phenomenon.

Mention should also be made of the so-called orthostatic tachycardia which was observed by Thomayer in three cases. It occurred in patients while standing and disappeared while lying down. The pulse rate varied between 76 to 90 and 168. Thomayer explains this tachycardia by asthenia of the cardiac motor centers in the oblongata. The forms of tachycardia occasioned by reflex action from the intestinal tract are characterized, as already stated, by a pulse rate rarely exceeding 160. Paralysis of the vagus is a part manifestation of various diseases, and is therefore combined with many other symptoms. In one case there are laryngeal disturbances, aphonia, and hoarseness;

in another, paroxysms of pertussis; in still another, dyspnœa without cyanosis; in others, considerable retardation of the dyspnœic respiration, and in others again, no retardation, but rather an increase of the frequency of respiration, due to limitation of the respiratory surface. A patient, whose vagus had been included in the ligation of the carotid, cried out that he was suffocating, his voice became hoarse, and when the nerve was touched there was vomiting.

Paroxysmal Tachycardia.—In contradistinction to symptomatic tachycardia, the group described as paroxysmal tachycardia exhibits an enormous frequency of heart-beats: 200, and even 300 per minute. The fundamental characteristics of this condition include, in addition to the excessive tachycardia, markedly lowered blood pressure, together with the peculiarity that the paroxysms commence, almost without exception, suddenly and disappear just as suddenly, as if with a jerk. The attacks may last for hours, days, weeks, and even months; they may end in death, but also—which is far more frequently the case—in complete recovery. The attacks are sometimes brought on by overexertion; also from the heart, by reflex action from the stomach and intestines, by the ingestion of sour or gas-forming food, or by the brain, through psychic excitement, fright, or terrifying dreams. A female patient under observation at the clinic dreamed that she was falling down a precipice; she awoke much frightened, and had a paroxysm which began with a stitch in the cardiac region. The paroxysm commences after prodromal signs, as vertigo, constriction of the thorax, epigastric pain, at times epistaxis; frequently, however, quite suddenly, without any premonition and introduced simply by a few irregular heart-beats. The pendular rhythm, with uniformity of cardiac pauses and equality of the first and second cardiac sounds in strength and accent, is explained by the enormous acceleration of the cardiac action. This pendular rhythm may occur as expression of cardiac insufficiency and sinking blood pressure in symptomatic tachycardia and in cases of moderate acceleration of pulse. In the tachycardia of Basedow's disease, where the blood pressure is, as a rule, normal or increased, the difference existing in strength and accent between the first and second cardiac sounds, as well as the relation between the short and long heart pauses, remains proportionate to the acceleration. The pendular rhythm in paroxysmal tachycardia, then, is the effect of the enormous acceleration of cardiac action and of low blood pressure. The cardiac impulse in paroxysmal tachycardia is either weak or distinctly visible and palpable, but even with energetic contraction of the heart the pulse remains weak and easily compressible. This contrast between the energy of cardiac contraction and reduction of the pulse tension distinguishes paroxysmal tachycardia from foetal heart rhythm (embryocardia), observed in the course of various infectious diseases, especially in grave typhoid. At times, however, during an attack of paroxysmal tachycardia, the pulse, hardly palpable at first, becomes more vigorous again in spite of its enormous acceleration, although the cardiac impulse does not show any additional energy. This points to a certain independence of the phenomena between heart and pulse. The pulse is subdicrotic, hyperdicrotic, at times monocrotic, always regular, and only at the end of the paroxysm is it irregular as at the beginning. Respiration is not disturbed, as a rule; the respiratory quiet marks a striking contrast to the stormy action of the heart, the face is rather pale than cyanotic, and there are no pulmonary râles, no emphysema. This is in contrast to a vagus neurosis, where frequently the pulmonary components, like asthma and acute distension of the lungs, make their appearance at the same time. In protracted paroxysms, in rare cases, even after a short time, there develop signs of cardiac inefficiency, stasis of the lungs, dyspnœa, cyanosis, engorgement of the intestines, ascites, edema, and dilatation of the heart. There are cases, however, in which such dilatations are not demonstrable either by percussion or the Roentgen picture. No parallelism exists between the cardiac dilatation and tachycardia, as the dilatation is not an integral part of the disease, but only a secondary manifestation.

Paroxysmal tachycardia, as a rule, is a painless affection, which distinguishes it from stenocardiac attacks. Sometimes, however, the paroxysm is introduced by a painful sensation in the cardiac region, constriction of the thorax, and even paraesthesia of the left arm; but in the course of the attacks these symptoms do not form a prominent part in the pathological picture. Diuresis in protracted paroxysms is nearly always reduced; sedimentum lateritium and albuminuria occur as signs of renal stasis. Huchard observed glycosuria, and polyuria with low specific gravity, such as urina spastica, has been described as occurring both before and after the attacks. Other nervous symptoms, insomnia, delirium, cephalalgia, gastralgia, articular pains, unequal size of pupils, myosis, mydriasis, profuse perspiration, especially disappear toward the end of the paroxysm, while rise of temperature to 39°or 40°, anorexia, coated tongue, vomiting, singultus,—accessory manifestations—may vanish in a moment after the attack is over. There are also paroxysms in which the patients do not experience any special subjective complaints. They follow their vocations and sleep quietly even during the attacks; their mental function may also be perfectly normal, a proof that ischemia of the brain cannot be as pronounced, as might be expected, judging by the smallness of the radial pulse.

ETIOLOGY OF PAROXYSMAL TACHYCARDIA.—The etiology of the disease is as yet entirely unknown. Paroxysmal tachycardia attacks individuals who have neither a hereditary, hysterical, neurasthenic, or epileptic predisposition. In some cases direct or indirect hereditary taint can be demonstrated. Burckland mentions the case of a girl suffering from tachycardia whose father had dementia paralytica and whose grandmother was mentally affected. Recently I saw a 16-year old student whose parents did not apparently offer any nervous manifestations, but the mother admitted that at the beginning of pregnancy she experienced a violent mental shock (partial loss of her fortune). In a few cases of infectious diseases, like typhoid and neuralgia, or overindulgence in coffee and tea, have preceded it. In one case paroxysmal tachycardia developed after belladonna poisoning, in another case after serious concussion of the thorax.

Paroxysmal tachycardia may also occur in individuals whose heart was not normal before the attack. This development of tachycardia on the soil of preexisting cardiac lesion is very frequent, so that one might almost be tempted to think of a more intimate connection between the two affections rather than of a mere coincidence. Several cases of this complication of organic affections of the heart with paroxysmal tachycardia have been in our clinic. One case was that of a 39-year-old tailor, whose pulse showed a frequency of 180 to 192 beats during the attack. The pulse was soft, dicrotic, but perfectly regular and uniform; the apex beat palpable as a systolic elevation in the fifth intercostal space, a finger's width outside the mammillary line; the first sound at the apex was accompanied by a short, blowing, systolic murmur, a pure second sound higher in pitch. The second sound at the base was pure, not accentuated. The tachycardia persisted until death. Autopsy disclosed complete adhesion of the pericardium to the heart, dilatation of a fatty heart, signs of congestion, mediastinum free from new tissue, vagus nerves intact. The short period of observation rendered it impossible to state with absolute certainty whether in this case there was a combination between the organic heart affection and a paroxysmal tachycardia. The lesson, however, to be drawn from this case is that the most pronounced acceleration of pulse may be present as the prominent symptom in the final phase of a pericardial adhesion.

Paroxysmal tachycardia may also occur as a complication of Basedow's disease, not always as an exaggeration of the accelerated pulse; in paroxysmal tachycardia, exophthalmus and enlargement of the thyroid gland have never been reported, nor has, on the other hand, such a fall of the blood pressure been observed in Basedow's disease as in paroxysmal tachycardia, all of which goes to prove that the two affections have no connection with each other.

The preceding observations demonstrate that paroxysmal tachycardia cannot be explained in these cases by paralysis of the pneumogastric nerve, because in the latter condition the accelerated pulse is permanent and never attains such an extreme height. Again, an irritation of the accelerans cannot last for weeks, aside from the fact that experimental irritation of the sympathicus does not cause such pronounced acceleration of cardiac action. The tachycardia occasioned by irritation of the accelerans also, as has been emphasized before, rises and disappears gradually. The myocarditis which has been found present in most cases at autopsy, often apparently of recent origin, can hardly be considered the cause of this condition. Symptomatic tachycardia certainly occurs in myocarditis, but generally the cardiac function is irregular, and there are other signs of cardiac inefficiency.

The theory of primary dilatation of the heart, as expounded by Martius, who looks upon tachycardia as a kind of vital compensation for the sacrifice of cardiac contractive energy, has not been accepted by the majority of authors. The dilatation of the heart cannot develop suddenly and as suddenly disappear, aside from the fact that in many cases it is not demonstrable either by percussion or by Roentgen rays. Even the vagus theory does not explain its origin. If a sudden interruption of the normal pneumogastric tonus is assumed to result from the influence of slight irritation of the oblongata—that is, a torpor of the inhibitive center, after the manner of hypnosis—no explanation is obtained of the extreme acceleration of pulse, which does not occur in anatomical lesions of the vagus, nor of the sinking of the blood pressure, which rises in lesions and paralysis of the vagus, as well as in atropin poisoning. If such an interference with the innervation in the central vagus nucleus is assumed, it is necessary to think of an irritation of the depressory fibers.

The difficulty of forming a judgment on the lowering of the blood pressure in paroxysmal tachycardia lies partly in the fact that we are not sufficiently informed on the conditions of pressure at the beginning and end of the attacks. The classical picture of paroxysmal tachycardia, with its sudden beginning and ending, suggests a neurogenic origin.

The lowered blood pressure, together with all the other frequent though not constant symptoms—polyuria, glycosuria, disturbance of pupils, migraine, perspiration—indicates a functional neurosis of the cardiac motor center in the oblongata. The most probable theory is that of Hoffmann, who likewise looks upon the bulbar cardiac motor center as the first point of attack in the development of the tachycardiac paroxysms. He assumes that by nervous influences which originate in the oblongata the excitability of the heart and its contractile and conductive power are sufficiently augmented to bring about more frequent cardiac irritations. In Hoffmann's case of paroxysmal tachycardia there was a duplication of the original rhythm and a distinct alteration between the greater and smaller pulse-beat. The study of the pulse curves also shows that the arhythmia observed in the intervals between the attacks is caused by extra systoles, which do not originate in the ventricles, but in the auricles, or the veins. This favors the assumption that "the same cause which leads to the extra systoles originating in the veins produces the complete attack of heart delirium by excessively exaggerated irritability, contractility, and conductivity."

Diagnosis of Paroxysmal Tachycardia.—The diagnosis of paroxysmal tachycardia is not particularly difficult in patients with whom the physician is personally acquainted, or where there was no preexisting organic affection of the nervous system or of the heart. The exceedingly high pulse rate, the dicrotism distinctly visible by sphygmographic tracing, and the absolute regularity of the pulse, with markedly lowered blood pressure, the sudden onset and the sudden cessation, as well as the repetition of the attacks, do not admit of any doubt as to the character of the disease. The diagnosis, however, becomes more difficult if paroxysmal tachycardia occurs in strangers, and especially in those who suffer from organic affections of the nervous system or of the heart, as was exemplified by two cases that occurred in our clinic. One of the cases, with pericarditic adhesions, has already been mentioned. The second was a female patient suffering from typical mitral stenosis with presystolic thrill, and the attack was complicated by pronounced cyanosis, venous pulse, congestion of the liver, ascites, and edema of the legs. Patient made the impression of one in the last stage of cardiac inefficiency, but the pulse showed no arhythmia, in spite of considerable frequency and the presence of mitral stenosis. Digitalis with analeptics (camphor, ether) proved useless. In a moment the pathological picture changed. The attack was over and the patient showed merely the signs of congestion, together with the well-known physical findings of mitral stenosis, and also an arhythmic pulse. Patient had a second similar attack, which lasted about two days and came to an end just at the moment when she was to be presented at a clinic. The manifestations of congestion disappeared under digitalis and the patient left the hospital improved.

TREATMENT OF PAROXYSMAL TACHYCARDIA.—There is no doubt that a number of cases, in spite of severe manifestations, recover or improve without treatment; but, on the other hand, there are individuals with heart disease in whom a short paroxysm may cause death, and even perfectly healthy subjects may share the same fate, if the paroxysms are prolonged and intense. Besides, paroxysmal tachycardia may be the prodromal symptom of a cardiac affection. Complications with other manifestations, as dilatation of a pupil without reflex action, ptosis, paralysis of the ocular muscles, and other signs of an organic nervous affection, also epilepsy and glycosuria, should be carefully considered in each individual case, as their presence would impose reserve as to prognosis. This shows the uncertainty in the prediction of most cases of paroxysmal tachycardia, although, generally speaking, short attacks in individuals with sound hearts take a favorable course. Wood's patient, who suffered from paroxysmal tachycardia for thirty-seven years, attained an age of 87; but, then, his attacks never lasted more than twenty-four to thirty-six hours.

The remedies recommended to terminate or shorten the attacks, as compression of the vagus at the neck, drinking of cold water, deep inspirations, hard pressing with closed glottis, sudden horizontal posture, low position of the head, tickling of the throat, and passage of sounds, emetics, digitalis, antipyrin, quinine, ergotine, belladonna, morphine injections, opium enemata, potassium bromide, ammonium bromide, caffein, spraying with methylchloride, laxatives, irrigations of the stomach, large doses of bicarbonate of soda in hyperacidity, venesection in congestion, leeches to the cardiac region, ammonium valerianat, ether—all these remedies are inefficient in the majority of patients, although they are apparently successful in individual cases. Ott's case, in which tachycardia disappeared when a peripheral arthritic attack came on, should, from a therapeutic standpoint, be attributed to a possible gouty diathesis. In other cases of symptomatic tachycardia, the treatment depends upon the nature of the original condition.

APPENDIX

I

THE CAUSE OF THE HEART-BEAT *

By W. H. Howell, M. D., Ph. D., Baltimore

Before William Harvey's time, physicians thought that the expansion or diastole was the active phenomenon of heart-beat. He demonstrated by experiments, however, that the pulse of the heart and arteries is due to a contraction of the cardiac musculature during systole, driving the blood forcibly into the arteries. His work marks the dividing line between ancient and modern theories. Harvey may also be regarded as the founder of the myogenic theory of the heart-beat. He interpreted systole and diastole as a contraction and relaxation of the musculature, and seemed to consider it due to an inherent property of the heart muscle itself.

On further analysis other writers developed hypotheses which attributed the principal cause of heart-beat to the nervous system.

Some were of Willis' opinion, namely, that the cerebellum was the seat of the stimulus. Borelli assumed that the nerves conveyed a liquid, a succus spirituosus which escapes slowly into the heart and sets up an

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* Abstract of papers in *The Journal of the American Medical Association*, June 2, 1906, p. 1665, and June 9, 1906, p. 1749.

ebullition, causing a contraction, or, according to his view, the expansion of systole.

In the eighteenth century Haller disproved apparently the neurogenic theory in the form it existed at the time and proposed a well-defined myogenic hypothesis, which marked the beginning of a discussion which has lasted until the present day.

He showed that the heart continues to beat after section of nerves leading to it, and as a result formed the hypothesis that the heart muscle possesses an inherent irritability or power of contraction which is independent of the influence of the extrinsic nerves. This, to his mind, was demonstrated by the fact that a quiescent heart may be excited to movement by heat, liquids, and electric sparks.

His theory, therefore, is that there is an inherent irritability of the heart muscle, and normally this property is aroused by the action of the blood flowing from auricle to ventricle.

In the nineteenth century, when physiology became more of an experimental science, this line of work was greatly aided by the advances made in the sister sciences of chemistry and physics.

The author states that we may well wonder why the capable observers of the time failed to discover the inhibitory action of the vagus nerve on the heart-beat, the demonstration of which is to-day so easy and successful. The explanation is evident, however, he remarks, when we consider the crude and inefficient methods of stimulation used at that time.

A new theory had meanwhile been devised. Legallois, as a result of experiments, concluded that destruction of the spinal cord is followed by failure in the power of the heart to maintain contractions.

We know that with modern technic such operations may be performed without causing death. Legallois was convinced that the principle of heart-beat is derived from the spinal cord in all of its parts, and is conveyed to the heart through the branches of the sympathetic system. Later experiments, however, soon made his conclusions improbable.

Throughout this period, during which efforts were made to show that the heart-beat is controlled directly from the central nervous system, physiologists were saved from obscure and doubtful theories by holding fast to the fundamental fact that the heart continues to beat after removal from the body. This fact, according to Volkmann, proves absolutely that the force causing the heart-beat does not arise in the brain or cord.

At this point the pendulum swings the other way and a newer theory appears. According to this view, the cause of the heart-beat lies in the automatic activity of the sympathetic ganglia present in the heart itself. Bichat divides the animal body into animal and organic processes. Over the latter, including the beat of the heart, the central nervous system has no control; they are under the influence of the sympathetic or ganglionic system. It was assumed that the sympathetic nerve consisted of a number of small independent systems, for each of which a ganglion served as a center or brain. This theory was strengthened in 1844, when Remak described nerve ganglia in the heart substance.

The neurogenic view in different phases was in favor until Gaskell, in 1881, advanced his arguments in favor of the myogenic theory. By a series of experiments on the rhythmic activity of the heart in frogs and tortoises, he became convinced of the fact that the heart muscle itself possesses the property of automatic rhythmicity, and that this property is most highly developed in the tissue at the venous end of the heart. He was led to believe that the systole begins at the venous end and spreads thence as a peristaltic wave over the rest of the heart, its rate being rapid in the auricles and ventricles and less rapid in the auricular-ventricular ring. The slower velocity at the latter point causes the apparent pause between the auricular and ventricular systoles. The nerve cells in the heart he considers as merely an inhibitory mechanism, and in no way the cause of the heart-beat.

Gaskell's ideas were supported by Engelmann, and from that time the myogenic theory has won many adherents. We know that normally the heart-beat begins in the great veins. The contractions of the remaining portions are due to impulses received from the venous end, either by way of the muscle or nerve fibers.

Considering the automaticity of the heart as a whole, it is clear that it depends for its initiation on the properties of that small portion of the musculature which forms the mouths of the great veins. The question arises whether the rest of the muscle of the heart possesses similar properties. Can any portion beat rhythmically if the connections with the venous end are severed?

Evidence shows that every portion, so far as the vertebrate heart is concerned, possesses in some degree or other the property of giving automatic rhythmic beats, provided the proper conditions are maintained. A portion of a heart removed from the organ may refuse to beat, even if supplied with normal blood, but if put under certain tension, or if certain substances are added to the circulating blood, it will give rhythmic contractions, showing that such portions of the heart possess a latent property of automatic rhythmicity which may be aroused if certain conditions are provided.

Therefore, if we could find a portion of the heart absolutely devoid of nervous tissue and so modify its conditions as to cause it to give beats of the usual cardiac type, we would have possession of facts that would seem to prove conclusively the myogenic theory.

The observation that the fetal heart begins to beat before it possesses nervous elements, is the only fact of this kind that remains unchallenged. In objection it has been urged that the fact that the beat of the embryonic heart is myogenic in origin is in itself no proof that the older heart, with its intrinsic nervous mechanism, continues to functionate in the same way.

Then, too, an improved technique may show histologically the presence of nerve elements in the embryonic heart.

The case at present gives strong support to the myogenic theory, but it has not sufficed to remove the objections of the other side.

Carlson, in his experiments on the heart of the limulus, has shown that not only the automatic beat of the heart but the conduction also of the wave of contraction is dependent on the action of the nerve cells in the median nerve cord. We have here, then, a case where the automatic contractions and the co-ordination of a genuine blood heart are undoubtedly of neurogenic origin.

We are not justified, however, in applying this result obtained in an invertebrate heart to the heart of vertebrate animals.

The cardiac muscle of vertebrates possesses peculiar properties which distinguishes it from the usual skeletal muscle. The most characteristic and fundamental of these properties is a long refractory period. That is to say: When cardiac muscle contracts spontaneously or as result of an artificial stimulus, it is entirely unirritable to further stimulation during the whole of its time of shortening. This reaction is the basis of the phenomenon of rhythmicity.

Under certain conditions the heart beat may be reversed, the wave of contraction beginning in the ventricles, passing on to the auricles and sinus venosus. This is compatible with a myogenic theory, but not so with the neurogenic hypothesis, for a system of connecting neurons is a mechanism that conducts and coordinates only in one direction.

In past years it was held that the myogenic theory was not applicable to mammalian hearts because no muscular fibers connect the auricles and ventricles. This, however, has been disproven in recent years by His, Tawara, and others who have shown that a small muscular strip passes from the auricle into the ventricular septum. Erlanger, especially, of the physiologists, has proved that the stimulus is conveyed from auricle to ventricle along this bundle of fibers. By compressing this bundle to a lesser or greater degree, he was able to break partially or completely the sequence of the ventricular on the auricular contractions. He also showed that in certain pathological conditions in man, a similar condition prevails, forming the important feature of the Adams-Stokes syndrome.

If the bundle was absolutely free of nerve fibers, the myogenic theory would be practically proven, but Tawara has shown that it possesses a net-work of nervous tissue like that lining the rest of the heart, so a positive answer to the problem is again postponed.

It has been shown that hearts of both warm and cold-blooded animals may be kept alive for hours after excision, if they are supplied with an artificial circulation. Hering was able to restore the beat for a period of several hours in an experiment on the heart of a man who had been dead eleven hours. He found that the inhibitory and accelerator nerves continued to give their respective effects for a long time. On the other hand, experiments on rabbits and dogs showed that sympathetic nerve ganglia lose their irritability very quickly after death, even when supplied with an artificial circulation.

It seems probable from these experiments that the maintenance of an automatic rhythm in the heart so long after excision or after somatic death can not be due to the activity of extrinsic ganglion cells. Since, moreover, the accelerator fibers retained their irritability for long periods after death, it would seem probable that they do not end in the ganglia of the heart, but are distributed rather directly to the heart muscles. This implies that the rhythm of the heart beat originates in automatic processes within the muscular tissue itself.

Whether we accept the neurogenic or myogenic theory, there remains for discussion the initial cause of the heart beat. Haller attributed this to the venous blood, acting as a chemical stimulus and exciting the heart. Other observers suggest that the blood acts simply as a mechanical stimulus to the heart, its pressure on the heart walls being the initial cause of the beat. Most physiologists during the nineteenth century took the stand that the blood has no direct influence on the production of the heart beat. The stimulus to beat arises within the heart itself as result of its own metabolism. There is no evidence except the automaticity itself that such a substance exists and no suggestion made as to its nature. If we accept this view we must assume that the unknown stimulating substance is a product of the resting metabolism of the heart. This view is advocated by Engelmann.

Most investigators have turned their attention to the careful examination of the individual constituents of the blood and the action of these on the heart muscle itself.

Ludwig and Merunowicz have studied the relations of the inorganic constituents of the blood to the heart beat. The latter was able to show that aqueous solutions of the ash of blood, free from all organic matter, had a marked effect in causing and maintaining the contraction of a frog's heart.

Ringer, in 1883, by accident was led to study the effect of calcium salts in the heart's contraction, and as result devised an artificial serum which bears his name and is capable of maintaining the beat of the heart in a wonderful way. This solution contains certain amounts of sodium, potassium, and calcium salts and for some hearts sodium carbonate. This serum has kept the hearts of certain animals beating for hours and even days. It must be understood that an aqueous solution of these salts together with oxygen cannot furnish nourishment to the heart in the sense of supplying it with a source of energy for its contractions. Their part must be found in their relations to the origination of the heart beat, that is to say, their connections with the chemical changes in the organic constituents which give rise to the energy of the contraction.

Experiments have shown that the potassium salts are not absolutely necessary as a constituent, but its presence does cause a normal beat for a longer time. Evidence shows that the potassium salts are concerned in the production of relaxation, diastole and inhibition. Therefore, in the matter of contraction, the sodium and calcium salts are of especial interest.

The author states that the following facts must be taken into account by any hypothesis which attempts to picture the processes causing the rhythmic contraction and dilatation of the heart muscle.

1. The heart possesses within itself a store of energy-yielding material, such that it may continue to give many hundreds or thousands of contractions after its supply of nutriment has been cut off.

2. Each contraction, whether caused normally or by an artificial stimulus, is maximal, and therefore, probably, uses up all of the energy-yielding material which is at that moment in an irritable condition, that is to say, in such a condition that it may be acted on by a stimulus.

3. The amount of this material in irritable form is nil during the phase of systole, but increases in amount throughout diastole. We know, for example, that if stimulated just at the beginning of diastole the heart muscle gives a small contraction and that the contraction, which may be obtained later by artificial stimulation, increases in extent the farther the diastole has progressed.

4. If the above statements are correct, it follows that the store of energy-yielding material in the heart

exists in some non-irritable form and that during the phase of diastole a portion is converted into an irritable form capable of being acted on by a stimulus.

5. The presence of certain inorganic salts is necessary for this transformation from the non-irritable to the irritable condition.

The hypothesis which the author has formed to guide him in his investigation is as follows: The well nourished heart contains a large supply of energy-yielding material, which is in stable form, so that it neither dissociates spontaneously, nor can be made to do so by the action of external stimuli. It is possible that this stable, non-dissociable form consists of a compound between it and the potassium or the potassium salts and that herein lies the functional importance of the large amount of potassium contained in the tissue. This compound reacts with the calcium or with the calcium and sodium salts, and a portion of the potassium is replaced and a compound is formed which is unstable.

At the end of the diastolic period, this compound reaches a condition of instability such that it dissociates spontaneously, giving rise to the chain of events that culminates in the normal systole. Before spontaneous dissociation occurs it may be hastened prematurely by an external stimulus, as we know to be the case when a mechanical or electrical shock is applied to the heart at any time after diastole has begun. This hypothesis accounts for some of the most characteristic features of heart beat.

Each contraction must be maximal since it involves the dissociation of all the material existing in unstable form. The contractions must be rhythmic since, after each contraction a certain interval, which will be constant when the conditions are uniform, is needed for the production of more of the unstable material. At each systole the heart will exhibit a refractory phase, since the ready formed, unstable material has been used up and the rest of the energy-yielding substance exists in a stable, non-irritable form. In terms of the hypothesis the refractory phase would pass off gradually as new, unstable material accumulates, and this we know to be the case since it requires a weaker stimulus to force the heart to contract the later it is applied in the diastole phase.

II

ADAMS-STOKES SYMPTOM COMPLEX.

ADAMS.* (*Dublin Hospital Reports,* 1827, Vol. IV, 396.)

" An officer in the revenue, aged 68 years, of a full habit of body, had, for a long time, been incapable of any exertion, as he was subject to oppression of his breathing and continued coughing.

" In May, 1819, in conjunction with his ordinary medical attendant, Mr. Duggan, I saw the gentleman. He was just then recovering from the effects of the apoplectic attack, which had suddenly seized him three days before. He was well enough to be about his house and even to go out. But he was oppressed by stupor, having a constant disposition to sleep, and still a troublesome cough. What most attracted my attention was the irregularity of his breathing, and remarkable slowness of the pulse, which generally ranged at the rate of thirty in a minute. Mr. Duggan informed me that he had been in almost continual attendance on this gentleman for the last seven years, and that during that period he had seen him, he is quite certain, in not less than twenty apoplectic attacks. Before each of them he was observed, for a day or two, heavy and lethargic, with loss of memory. He would then fall down in a state of complete insensibility, and was on several occasions hurt by the fall. When they attacked him, his pulse would become even slower than usual, his breathing loudly stertorous. He was bled without loss of time and the most active purgative medicines were exhibited. As a preventative, a large issue was inserted in the neck and a spare regimen was directed for him. He recovered from these attacks without any paralysis. Edema of the feet and ankles came on early in December; his cough became more urgent and his breathing more oppressed; his faculties, too, became weaker.

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* As a matter of historical interest, it being the first recorded case presenting the symptoms of bradycardia associated with syncopal attacks, the original paper by Adams, published in *The Dublin Hospital Reports,* 1827, Vol. IV., p. 396, is here given verbatim with the findings at autopsy.

"November 4, 1819, he was suddenly seized with an apoplectic attack, which in two hours carried him off, before the arrival of his medical attendant.

"Dissection. The right auricle of the heart was much dilated. The right ventricle externally presented no appearance whatever of muscular fibers; it seemed composed of fat through almost its entire substance, of the same deep yellow color as that which occupied the place of the left lung. The reticulated lining of the ventricle, which here and there allowed the fat to appear between its fibers, alone presented any appearance of muscular structure.

"The left ventricle was very thin and its whole surface was covered with a layer of fat. Beneath this the muscular structure was not a line in thickness; it had degenerated from its natural state; was soft and easily torn, and a section of it exhibited more the appearance of liver than of heart.

"The septum of the ventricles presented the same appearance. In both ventricles, even in the lining fibers, yellow spots, where fat had occupied the place of muscular structure, were to be observed. The whole organ was remarkably light; the valves were all sound, except those of the aorta, which were studded with specks of bone, but elsewhere were cartilaginous and elastic, from which they derived a disposition to remain closed; a fluid gently injected from the ventricle would pass them; still, when the heart was reversed and water poured from the ventricle upon them, these valves retained it. Its weight was not sufficient to separate the edges of the thickened valves. There was much fluid blood contained in the heart."

STOKES. (*Dublin Quarterly Journal of Medical Science,* 1846. II, 73.)

The author's first case presented symptoms of repeated pseudo-apoplectic attacks not followed by paralysis, a slow pulse and a valvular heart murmur. This patient could at times ward off attacks by getting on his hands and knees and keeping his head low. The author also notes the pulsation in the right jugular vein, and states that the number of pulsations was "more than double the number of the manifest ventricular contractions."

Stokes' second case was that of a patient with a marked anemia. There was a very slow pulse and a valvular murmur. Death occurred apparently from syncope.

HIS.* (*Deutsch. Archiv. f. Klin. Med.*, 1899, B. 64, p. 316.)

Describes a case of Adams-Stokes disease with unequal contractions of the auricle and ventricle (heart block), which is interesting not only on account of the rarity of the disease, but also because of a phenomenon which, though well known in the physiology of animals, has simply been observed in man. It has not yet been fully described, and the recent work on the physiology of the heart promises to furnish its explanation.

The following is the case history:

G. G., a handworker, fifty-four years of age, was admitted on May 12, 1893, into St. Jacob's Hospital.

He had no hereditary predisposition. In childhood he had several of the diseases of that period, and about twelve he recovered completely from an inflammation of the lungs. Otherwise he was strong and healthy up to the beginning of his present illness.

He was married and had had five children, of whom three had died respectively at an early age of measles, diphtheria, and whooping cough. The last pregnancy of his wife ended at the third month in a miscarriage which followed unusual physical work. No history of lues was determinable. The present illness began on June 24, 1892. While engaged in heavy work in a freight station at night, things suddenly became blank before his eyes, he fell down unconscious and struck his face and head. After a short time he came to and found his way home alone. He remained quietly at home for a few days. On the fourth night after this attack he fell back while at closet, became suddenly nauseated, deathly pale, and unconscious. There were also some contractions of the face. This attack passed over quickly. The patient rested for five weeks, undertook only light work, and soon felt entirely well. In September he again took up his regular work, and during the winter had only two short attacks, each time after especially hard work. Severe attacks of the same kind on the 13th and 25th of April and on the 8th of March, and from this time on the attacks of unconsciousness repeated themselves at short intervals, especially after mental excitement.

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* His, whose experimental work on the heart, especially the production of heart block on animals, was very extensive, had the good fortune to observe this very typical case, the symptoms of which convinced him that he had to do with the same lesion that he had produced experimentally in lower animals.

Physical condition—Moderate emphysema. Lower border of liver two finger breadths below the border of the ribs, smooth and somewhat resistant. Cardiac dullness increased to the left. Apex beat neither visible nor palpable. Cardiac sounds are very soft but audible when the breath is held. The sounds over the apex are peculiarly modified but not blowing, softer over the tucuspid area and not audible over the base.

The temporal arteries are calcified and markedly serpentine. The radials are also calcified. The pulse on all arteries ranges from 36 to 44 per minute, moderately large, and has the character of a senile pulse.

On the second day (May 14) the pulse sank quickly from 36 to 18 without any preceding bad feeling or fear, then rose to 36, fell again to 12, and increased once more to 24. There suddenly occurred a characteristic paroxysm. Face and extremities showed epileptiform contractions, unconsciousness, respirations labored and dyspnœic, the face had a dark cyanotic hue, and the pupils were markedly dilated. This paroxysm lasted one and one-half to two minutes, passed off without leaving a trace, and recurred twice on the same day. The pulse was very small the entire day, varied in frequence between 24 and 60, and was at times regular and at other times irregular.

On May 15, during the morning examination, the pulse suddenly stopped and there occurred an epileptiform attack. There were clonic contractions of the mouth, the face, and the extremities. The patient became pale as death, and lost consciousness. The pupils were dilated and the bowels moved. After 23 seconds a pulse-beat was felt, then a second one, and they continued with increasing frequency, consciousness returned, the contractions ceased, the face took on its normal color, and the pupils gradually contracted. These attacks were repeated that entire day and the following night, often at intervals of only two to three minutes. The pulse varied in its frequency in the intervals, and at times was markedly bigeminus. On May 16 urine was voided for the first time. It contained a trace of albumen and many small hyaline casts. The attacks continued as on the previous day. In the intervals the pulse was regular, 20-30 beats per minute. A new and characteristic phenomenon was observed over the heart.

During the attacks when the breathing stopped and the pulse could not be felt at radial or carotid and the heart sounds at the apex were muffled, there was a very soft, irregular murmur appreciable at the base of the heart in the second and third intercostal spaces to the right and left of the sternum. Its frequence could not be exactly determined, but it was more frequent than the returning arterial pulsation. In the following two days the condition was practically unchanged. On May 20th the attacks did not occur as often, and at night took on a new characteristic. Pulse and respiration ceased at the same time, and associated with cyanosis and deviation of the eyes. After half a minute there was a respiratory gasp, which was followed by several pulsations, then in about seventy seconds another gasp with four or five small quick beats, and so it continued for five minutes. Finally the pulse and respirations were again continuous. The pulse was at first 60, after two minutes 36, then 24. The patient continued irrational, did not recognize his wife, and passed a restless night. On the evening of May 21st the paroxysms showed a new characteristic. After the pulse and respirations ceased, a regular Cheyne-Stokes respiration followed. When the first pulse-beat occurred the respiration ceased completely, the face became red, and then the contractions at the mouth, face, and extremities first appeared. A further phenomenon was observed at the same time. At the time when the pulse and respiration ceased, the cervical veins were well filled and manifested evident undulatory pulsations. These were apparently synchronous with the murmurs previously described over the base. Their frequence during and between the attacks was apparently constant, and was about 26 in a quarter of a minute. Even after the reappearance of the arterial pulse the venous pulse continued as a true negative venous pulse, modified, however, so that a collapse occurred after every third or fourth beat, which was then synchronous with the carotid pulsation. This condition continued until May 24th, when the attacks presented a new type. They began with cessation of the respiration while the pulse continued to beat regularly. Then the pulse stopped and the breathing assumed the Cheyne-Stokes character. Consciousness disappeared quickly with the cessation of the pulse, and returned with the reappearance of the pulse, together with marked erythema and profuse perspiration. Between these there were other attacks in which the pulse beat regularly and the breathing had the Cheyne-Stokes character. The condition improved gradually, the attacks were shorter and less frequent. The patient felt better and ate with relish. The venous pulse in the neck continued in frequence three to four times the arterial pulse, which ranged from 20 to 40, usually about 28. This improvement continued up to June

21st, when the attacks became more frequent and had the same character as when first described. Patient at the request of his wife left the hospital on June 27th.

He died three months later, but in spite of all efforts to obtain an autopsy, permission was not granted. It was unfortunate that an autopsy was not permitted in this typical case. The clinical symptoms, however, were so characteristic as to make the author's explanation beyond question.

In animals the heart-block has been known to every physiologist. The heart of every dying animal very commonly manifests it, and it can be artificialy produced with great ease. A ligature or pressure in the atrio-ventricular grove, injury to the cardiac musculature, an increased excitability of the auricle or of the venous sinus, finally under certain conditions the irritation of the vagus may be sufficient to induce it.*

There exists in men as well as in mammals a connective tissue layer between the muscle wall of the auricle and that of the ventricle, and this completely separates them. In this layer muscular fibers† have been found, and His has himself demonstrated that such a bundle is present in mammals, and extends from the posterior wall of the right auricle to the septum.

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* Engelmann: *Pflüger's Archiv.* 62, 1896; *Pflüger's Archiv.* 65, 1897. Kronecker: *Zeitschr. für Biologie,* 1897. "On the Innervation of the Heart," *Journal of Physiology,* Vol. IV. Wenckebach: *Zeitschr. für Klin. Med.,* 36, 1899. Tegerstedt; *Physiologie des Kreislaufs,* 1893, p. 246. Hunt and Harrington: *Journal of Exper. Medicine,* Vol. II., p. 723.

†Stanley, Kent, *Journal of Physiology,* XIV.

Experiments which he made with Graupner showed that when this bundle was destroyed, auricle and ventricle contracted at unequal times and without reference to each other.‡

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‡The curves and preparations of these experiments were demonstrated at the International Physiological Congress in Bern, 1896.

III

ADAMS-STOKES DISEASE.

ABSTRACTS FROM AMERICAN AND BRITISH MEDICAL LITERATURE AND FOREIGN BIBLIOGRAPHY.

GASKELL. (*Journal Physiology,* 1883. IV, 43.)

First showed in experiments on the hearts of tortoises that the auricles and ventricles beat independently of each other when the musculature of the auriculo ventricular junction is compressed.

OSLER. (*Lancet,* August 22, 1903. p. 516.)

Classifies cases of Adams-Stokes disease under three groups:

1. Post febrile. 2. Neuritic. 3. Arterio-sclerotic.

Nearly all his cases come under group 3. He gives an account of fourteen cases.

In discussing the jugular pulsation he says: "We cannot dismiss the question of independent auricular contraction as settled."

Discussing the pathology, he places cases of bradycardia under two groups.

1. The result of heart lesions. 2. The result of disturbances of the nervous system.

His opinion at the time was that the essential cause of the disturbance of rhythm was unknown.

NORFLEET. (*Medical Record,* November 21, 1903.)

Reports a case in a man 69 years of age, whose pulse for over a year never registered above 45 beats to the minute. During exacerbations, the pulse fell as low as 8 or 9 beats a minute, lasting twenty-four hours at a time. On such occasions there were convulsive movements.

KIDD. (*Lancet,* February 13, 1904.)

The author gives a case and then reviews the literature, giving the different theories as to the causation. He never examined the heart during attacks, but between attacks. The pulse and heart sounds corresponded exactly. There were no signs of general arterial degeneration. The patient had an aortic stenotic murmur.

LASLETT. (*Lancet,* June 4, 1904.)

Gives a case which is typical. Here the fit was usually brought about by some emotional disturbance. He suggests that from that fact we may suppose the slow heart beat due to the inhibitory action of the vagus.

GILLIES. (*Montreal Medical Journal,* June, 1904. p. 422.)

Gives an abstract on three cases by His, Jr., A. Hoffman, and A. Jacquet, but forms no conclusion as to the pathology.

PITTSFIELD. (*Medicine,* September, 1904. p. 654.)

Describes the disease and gives a case presenting the usual symptoms of Adams-Stokes disease and quotes the neurogenic theory of heart stimulus as a possible basis of the pathology of the disease.

HANFORD. (*British Medical Journal,* December 31, 1904.)

The autopsy findings in the author's case showed gummatous involvement of the bundle of His.

GOSSAGE. (*Transactions of the Clinical Society,* London, May 26, 1905, Vol. 39, p. 187.)

Describes Adams-Stokes disease as a collection of symptoms due to a number of pathological causes and notes the frequently associated arterio sclerosis in the majority of cases. After the specific fevers the degeneration of the heart muscle may be a cause in its production.

STODDARD. (*Wisconsin Medical Journal,* May, 1905. p. 669.)

Does not think there is evidence to prove that a permanent bradycardia can be caused by stimulation or irritation of the vagus.

He thinks the essential feature is the bradycardia and that this, affecting the cerebral circulation with the resulting alteration of blood pressure, results in the other symptons. He presents a case.

DOCK. (*Medical News,* August 19, 1905.)

Reports a case in which the ventricular contractions were not affected by the administration of atropine, showing that the pneumogastric has no effect on the ventricle if stimuli are unable to cross over the bundle of His.

FOLEY. (*Boston Medical and Surgical Journal,* August 31, 1905, p. 235.)

Presents two cases of his own and gives a resume of the literature, especially with reference to the pathological findings.

MAYNARD. (*British Medical Journal,* October 7, 1905.)

Assumed that there was a dissociation of the two ventricular beats because of the absence from the descending or diastolic limb of the sphygmogram of any indications of the precordially well marked, audible and palpable secondary impulses. Others, however, thought that the unregistered impulses might be due to auricular pulsations.

MOON. (*Transactions of Clinical Society of London,* October 27, 1905, Vol. 39, p. 214.)

Gives a typical case.

MORRISON. (*British Medical Journal,* November 11, 1905.)

Discusses the heart sounds heard during the long diastolic pause observed in Adams-Stokes disease. He thinks it is due neither to an abortive systole of the left ventricle or an auricular systole. His opinion is that it is best explained as a mode of hemisystole of the right ventricle. He states that the myogenic theory in cardiac action has been made too much of at the expense of the haemic and neural factors and thinks heart block is still only a theory.

ERLANGER. (*Journal of Experimental Medicine,* November 25, 1905, p. 676.)

The author presents a case of Adams-Stokes disease from which, by very complete experiments with cardiogram, sphygmogram, etc., he finally summarizes as follows, viz.:

The symptoms of Adams-Stokes disease are caused by the same lesion which causes heart block.

When the block is complete, stimulation of the vagus causes the auricles to beat but not the ventricles. When the block is partial, the rate of ventricular contractions varies proportionately with the rate of auricular contractions, but only within certain limits.

STENGEL. (*Americal Journal of Medical Science,* December, 1905, p. 1083.)

Defines and discusses heart block and the myogenic theory of heart stimulus. He states that this theory is the most convincing in view of recent experiments, which have shown that the symptoms of experimental heart block and Adams-Stokes disease are identical. His case shows pathologically a lesion involving the bundle of His.

He describes his own case very fully. In this case there were no sounds heard over the praecordia suggesting venous pulsation. For the last few days before his patient died a condition of complete block existed, the radial pulse ranging from 18 to 26 beats per minute, while the venous pulse in the neck registered from 80 to 140 beats per minute.

On autopsy he found an atheromatous patch on the anterior mitral leaflet toward its base and aortic edge. This extended to the endocardium exactly over the bundle of His, where the bundle passes from auricle to ventricle. On the left ventricular side it was evident that the bundle of His was involved.

RITCHIE. (*Proc. Royal Soc. Edin.,* 1905. XXV, 1085.)

Demonstrated by fluoroscopic examinations that the auricles and ventricles beat separately.

ERLANGER. HIRCHFELDER. (*American Journal of Physiology,* January 1, 1906, p. 153.)

The authors give a summary of previous experiments on heart block and then describe experiments on twenty-one animals using the heart clamp to bring about heart block by pressure on the bundle of His.

They showed definitely that a temporary stoppage of the ventricles can be brought about by four different methods:

1. By blocking mechanically the passage of an impulse through the bundle of His.
2. By stimulating the vagus center.
3. By a sudden stopping of rhythmic stimulation of the ventricles in complete block.
4. By increasing the auricular rate during partial block.

HAY. (*Lancet*, January 20, 1906, p. 139.)

Discusses the characteristics of cardiac muscle and gives its five great functions.

1. Rhythmicity. 2. Excitability. 3. Conductivity. 4. Contractility. 5. Tonicity.

He states that the heart block in his case was due to a depression of conductivity which later became normal. Still later a condition of heart block existed due to a depression of the function of excitability. Heart sounds were heard during ventricular diastole, caused, in his opinion, by the systole of the right auricle.

He thought the depression of conductivity and excitability of the myocardium due to impaired metabolism, secondary to an insufficient blood supply, the result of arterio-sclerosis of the coronaries.

The excitability of the musculature was not stable, possibly as result of nervous influences such as mental excitement.

GRÜNBAUM. (*British Medical Journal,* February 10, 1906, p. 378.)

Showed a gummatous involvement of the interventricular septum and bundle of His.

WILLARD. (*British Medical Journal,* February 10, 1906, p. 378.)

Gives an interesting account of bradycardia following head injury to a man 64 years old. Previously the patient's health had been good. On entrance his pulse was 56, but two days later it dropped to 28, and on the seventh day to 23. His arteries were atheromatous and there was no valvular disease, but all the heart sounds were weak, and the sounds agreed with the radial pulse. He states that the cause of inhibition of the heart's action is difficult to explain. There was evidently an interference with the pneumogastric or sympathetic, but where the interference occurred is uncertain.

FINNY. (*British Medical Journal,* April 28, 1906. p. 967.)

Discusses briefly the myogenic theory. Uses the term heart block as applied to the failure of an impulse, originating in the muscles of the auricles, to pass the auriculo-ventricular septum through the bundle of His and to stimulate a contraction of the muscles of the ventricles.

His case is a woman with pulse about 22 per minute. The jugulars pulsated two or three times to each ventricular systole. X-ray showed the auricles beating between the ventricular contraction.

There were also pauses lasting for some seconds when no radial pulse was felt, during which time she had epileptiform attacks. For the first few days prior to death, these periods would last as long as seven minutes.

He mentions the Adams-Stokes syndrome as associated with advanced age and arterio-sclerosis, but in this case the woman was only 42 with no apparent organic disease.

In connection with the arhythmia were heard faint heart sounds in the intervals between the regular systolic ventricular contraction.

He says these sounds may be due either to:

(1) A hemisystole of the heart, *i. e.*, the right ventricle contracting while the left does not.

(2) A rhythmic contraction of the auricles acting alone, or,

(3) A feeble ventricular contraction.

In this case he thinks the arhythmia and not the bradycardia the cause of the convulsions.

There was no autopsy.

GORDINIER. (*Albany Medical Annals,* June, 1906.)

After discussing the etiology, pathology, and symptomatology, presents three cases of Adams-Stokes disease

CHAPMAN. (*Lancet,* July 28, 1906. p. 219.)

Gives a case of bradycardia due to cardiac syphiloma in which the heart showed marked fibrous changes. The pulse varied from 33 to 44, but there were no syncopal attacks. The patient died later of peritonitis following appendicitis.

Besides the fibrous changes in the myocardium, there were calcareous deposits in the endocardium and the coronaries were narrowed at their orifices. There was also atheroma of the aorta. He asks in this case, was the bradycardia due to myocardial invasion with a gummatous deposit and subsequent fibrous changes, or was it due to an early implication of the orifices of the coronary arteries?

STEINER. (*Boston Medical and Surgical Journal,* August 9, 1906, p. 135.)

Gives a report of three cases. He discusses the etiology and pathology and thinks the general trend of opinion is in favor of the myogenic theory of heart beat rather than the neurogenic. He describes heart block partial and complete and describes Erlanger's experiments in heart block on animals. His opinion is that the interposed heart beats are due to auricular systole.

ASCHOFF. (*British Medical Journal*, October 27, 1906, p. 1103.)

Presents in detail the gross and minute anatomy of the heart and heart muscle, especially with relation to the bundle of His.

In one of his two cases of Adams-Stokes disease he found a marked sclerosis of the small arteries which course through the bundle, and in the other case a marked development of adipose tissue in the same place.

MACKENZIE. (*British Medical Journal,* October 27, 1906, p. 1103.)

Defines heart block as a condition where, on account of some defect in the muscle fibers joining the auricles and ventricles, the stimulus for contraction passing from auricle to ventricle by these muscular fibers is stopped. The main function of these muscle fibers is to convey the stimulus of contraction from the auricle to the ventricle.

The depression may be so marked that the stimulus does not reach the ventricle, which does not contract with the auricle and a systole is dropped out.

The syncope in Adams-Stokes disease depends on the slowing of the ventricular contractions.

ERLANGER. (*British Medical Journal,* October 27, 1906, p. 1103.)

The author demonstrates how pressure in the bundle of His retards stimuli and section or destruction of the bundle results in a complete and permanent heart block.

The ventricles possess a dormant rhythmicity, and when the block is complete, the ventricles, instead of following the auricles, beat independently, at first very slowly, but later on increase in frequency until a constant but still slow rate is maintained.

The apoplectiform attacks are due to venous congestion, while the epileptiform seizures are due to a cerebral anemia.

GIBSON. (*British Medical Journal,* October 27, 1906, p. 1103.)

Suggests that the great difference in venous pulse and arterial pulse may be due to an arrest of impulse at the sinus as well as at the auriculo ventricular band.

He is of the opinion that the faint heart sounds heard in the interval between ventricular pulsation are due to auricular pulsations as they coincide with the venous tracings which are attributed to the contractions of the auricles.

His case showed increase in fibrous tissue with wide separation of the muscle fibers of the bundle of His.

MORROW. (*British Medical Journal,* October 27, 1906, p. 1103.)

Presents a case of arhythmia due to a failure of contractility. The contractility of the heart muscle was lost, he thinks, due to a too frequent rate of heart beat.

BARR. (*British Medical Journal,* October 27, 1906, p. 1122.)

Describes his case where in periods of unconsciousness the pulse fell as low as eight beats to the minute. Autopsy showed dilated and hypertrophied heart, thickening of the endocardium at the valve cusps and some thickening of the coronary arteries. He does not think that the theory of the degeneration of the bundle of His explains the phenomena of the disease and is doubtful that impulses are normally sent from auricle to ventricle through the bundle of His. He considers the so-called myogenic theory merely an inference. The author thinks that it is not sufficiently well recognized that the heart is not a single organ but is really two organs, each with its own definite work to perform. He admits that all parts of the heart work rhythmically, but even in perfect health the rhythm is subject to variation. He is open to conviction but is pleased to hope and think that the activities of the ventricles do not hang by such a narrow thread.

SCHMOLL. (*Journal A. M. A.*, 1906. XLVI, p. 361.)

In the author's case, atropine given had no effect on the ventricular beat, but the auricular pulsation was reduced from twelve-tenths to eight and one-half tenths of a second.

JELLICK. COOPER. OPHULS. (*Journal A. M. A.*, LXVI, p. 955.)

Present a case of epididymitis, probably gonhorrheal, which went on to septicemia, during which the Adams-Stokes syndrome made its appearance fourteen days before death. The autopsy showed anemic necrosis of the muscular septum in the region of the bundle of His as the result of a recent thrombosis of its nutrient artery. The heart was otherwise healthy.

KEITH and MILLER. (*Lancet,* November 24, 1906.)

Report a case of marked gummatous deposit in the bundle of His.

KAHN. (*Journal A. M. A.,* December, 1906, p. 2011.)

The author has noted a number of cases of bradycardia in gangrenous appendicitis. In his opinion the cause is probably the absorption of ptomaines, which during circulation act on the cardiac ganglia.

WILLIAM EWART. (*Progressive Medicine,* 1906.)

In his section on heart block and Adams-Stokes affection, he gives a review of the literature on the subject, beginning with His, Jr., article in 1893, and reviewing up to the time of the presentation of his article.

ASHTON. NORRIS. LAVENSON. (*American Journal of Medical Science,* January, 1907.)

The authors record the histological findings of a case of complete heart block in which they found a gummatous deposit that involved the bundle of His. The muscular fibers were entirely replaced by the gummatous mass, so that the continuity of the bundle was absolutely destroyed, resulting in complete block.

BUTLER. (*American Journal of Medical Science,* January, 1907, p. 715.)

Gives a typical case of complete block. Microscopically there was found a fatty infiltration of the bundle of His, so marked that the bundle was about one-fifth of its normal size.

DOCK. (*British Medical Journal,* October 19, 1907, p. 1039.)

Discusses disturbances of rhythm in heart disease. In his section on heart block he gives a case of his own where on autopsy there were found fibroid changes in the endocardium over the left branch of the bundle of His and also in the branch itself. There was also an endarteritis of the nutrient artery of the branch. He thinks that in this case there was a partial block due to an ischemia, increased at times by spasm of the artery. Future examinations of sections may reveal important lesions.

BEARDS. (*Detroit Medical Journal,* October, 1907, Vol. VII, No. 10.)

Gives a typical case of complete block, with Mackenzie's polygraph tracings. The jugular pulsation was 72 while the apex beat, radial and carotids beat 24 to the minute or a 3 to 1 ratio.

On strychnine the pulse went up to 35 and remained so. Syncopal attacks have also diminished.

HOUSE. (*Journal A. M. A.*, Nov. 30, 1907, p. 1839.)

The author is of the opinion that the myogenic theory, as shown by the experiments in artificial heart block, does not explain certain cerebral symptoms which occur in Adams-Stokes disease. A lesion of the heart alone leaves too much to the imagination. Despite post-mortem findings, he feels that to consider the lesion of the bundle of His alone the cause of the disease, without considering an involvement of its nerve filaments, is to consider the myogenic theory proven conclusively, and this he feels is not definitely proven.

The neurologists have a basis for their opinion that the lesion is one of the central nervous system when we consider that the cardiac and respiratory centers are known to be located in the floor of the fourth ventricle. He thinks that the possibility of the lesion being in the central nervous system has been slighted, considering the many nervous symptoms shown in cases. The pathology is not yet established.

In the reported case of the author may be noted of special interest: increased patellar reflexes and unequal pupils with what was regarded as an arterio-venous aneurism of the right disk.

The diagnosis was that of a new growth, possibly an aneurism about the pons, irritating the right vagus. Sarcoma and gumma were also considered possible. Of interest also is the fact that the disease has existed for eight years, and the patient is as well now certainly as he was two years ago.

As the disturbance of reflexes can only be explained by a lesion of the central nervous system, the author concludes that he is justified in believing that the lesion was of the tenth nerve at certain points in its course.

IV

TACHYCARDIA

ABSTRACTS FROM AMERICAN MEDICAL LITERATURE AND FOREIGN BIBLIOGRAPHY

MORRISSEY. (*Medical Record,* December 2, 1905.)

Gives conditions where a rapid pulse is found. He states that some cases are due to myocardial degeneration and profound shocks to the nervous system, but when it comes to the pathology of tachycardia, the author places it in the region of theoretical speculation.

WARDEN. (*Journal A. M. A.,* January, 1906.)

Does not think paroxysmal tachycardia a definite entity. He gives an account of two cases in which, during the paroxysm, the pulse rate was respectively two and three times faster than normal.

HEWLETT. (*Journal A. M. A.,* March, 1906, p. 941.)

In his article on "The Doubling of the Cardiac Rhythm and its Relation to Paroxysmal Tachycardia," states that no physiological explanation of this form of tachycardia has been given. It suggests a neurosis, yet experimentally it cannot be produced.

In many cases an abrupt doubling of the cardiac beat at the beginning of tachycardia is followed by a return to normal as abrupt as the onset.

In the author's case, at times the radial pulse was only one-half that of the cardiac pulsation. During attacks, however, the pulse rate was often double that between attacks.

He advances Hoffmann's theory, namely: that the heart is stimulated much more frequently than its rate would indicate. The failure to respond to every stimulus may be accounted for by Engelmann's hypothesis, namely, the irritability of the heart muscle is lessened after a contraction and may not recover from one stimulus in time to respond to the next one. He suggests the heart block theory also.

In conclusion, he states, that paroxysmal tachycardia, whether a definite disease or a symptom complex, is yet undecided. Many cases seem to rest on a neurotic basis.

On the other hand, coronary sclerosis may be present and it can seldom be excluded with any degree of certainty.

In his case the man showed evidence of myocardial disease. The author thinks it may be merely a symptom that may be produced by different causes and anatomic change in the cardiac muscle may or may not be present.

HIRSCHFELDER. (*Johns Hopkins Bulletin,* October, 1906.)

The author presents a case which showed the relation of two to one in the pulse, that is, rising from 80 to 160 during an attack. It also dropped immediately when the attack was over to 80. This fact makes him favor Hoffmann's theory that the periods between attacks are due to dropping of alternate impulses. In this case he does not think the block is at the auriculo-ventricular bundle, but at the sino-auricular junction.

He inclines rather to Hoffman's later theory, that the heart responds to all the impulses arising at the sinus only during the period of tachycardia; that between attacks, owing to a diminution in conductivity, which he refers to the sino-auricular region, the auricles and ventricles respond to only alternate or sometimes even only every fourth impulse from the sinus. The latter he thinks due to a depression of conductivity in two places.

WEINSTEIN. *New York Medical Journal,* Jan. 19, 1907.

The author, after discussing the cause and symptoms of tachycardia, presents a case, in a woman with marked gastroptosis. The use of an abdominal supporter soon brought the pulse down to normal.

His opinion is that the prolapsed stomach dragging on the vagi interfered with their functionating and so caused the tachycardia.

SCHMOLL. (*American Journal Medical Sciences,* November, 1907.)

Shows that in paroxysmal tachycardia we are dealing not with a single pathological entity, but with different pathogenic forms.

He divides his nine cases into four etiological groups:

1. Cases occurring in patients with previously damaged heart.

2. Cases occurring in patients affected by dysthyreosis.

3. Cases occurring in patients exhibiting central nervous lesions.

4. Cases apparently functional in character or idiopathic.

Five cases are presented, three of which come under heading No. 1 and two which come under No. 3 and No. 4 respectively.

An interesting feature of the first three cases is that the heart condition alone did not bring on the tachycardia. A febrile attack, mental strain and a cholecystitis respectively, brought on the tachycardia in these cases, acting as an extra weakening influence. When the heart muscle recovers, the tendency to attacks lessens.

In the author's fourth case, the lesion was probably due to sclerotic change in the basilar artery.

There were no signs of organic disease and very slight arterio-sclerosis in the fifth case. So the author places it under the idiopathic type.

From his study of tracings taken during normal and abnormal heart action, he distinguishes two forms of tachycardia:

(1) Forms in which the auricular wave is missing, the jugular pulse being of the ventricular form.

(2) Forms in which an auricular wave appears in the tracing obtained during the tachycardiac rhythm.

In these cases the ventricular systole follows an auricular contraction. From venous and arterial tracings he is convinced that the tachycardia is caused by extra systoles.

In conclusion he emphasizes the pathological importance of the artrioventricular conducting system.

VI

ADAMS-STOKES DISEASE

FOREIGN BIBLIOGRAPHY

Heine.—*Müller's Archiv.*, 1841.

Leyden.—*Virchow's Archiv.*, 1868, 44.

Cornil.—*Gaz. des hôp.*, 1875, No. 64.

Thanhoffer.—*Centralbl. f. d. Med. Wissensch.*, 1875, p. 405.

Federici.—*Rivista clin. di Bologna*, 1875, Feb. and March.

Korcziniski.—*Centralbl. f. klin. Med.*, 1877.

Riegel.—*Deutsch. Archiv.*, 1880, 27.

Samuelson.—*Zeitschr. f. klin. Med.*, Vol. 2, 1881.

Polak.—*Medycyna*, 1882, No. 51.

Stackler.—*Revue de Med.*, 1882, II.

Figuet.—*Etude sur le rhythme couple du cœur*, Thèse, Lyon, 1882.

Tripier.—*Revue de med.*, 1883, p. 1001.

Chauveau.—*Rev. de med.*, 1885, v. p. 161.

Grob.—*Deutsch. Archiv f. klin. Med.*, XLII., 1888, p. 574.

Germain See.—*Traité des maladies du cœur*, 1889, p. 523.

Regnard.—Etude sur le pathologie der pouls lent permanent. Thèse, Paris, 1890.

Sendler.—*Centralbl. f. klin. Med.*, 1892, p. 642.

Dehio.—*St. Petersburg Medicin. Wochen.*, 1892, No. 2.

Riegel.—*Volkmann's Samml. klin. Vortr.*, No. 227.

STRUBING.—*Deutsch. med. Woch.*, 1893.

ARNSTEIN AND EDGGREN.—*Gaz. lekarske*, 1894, No. 24.

HIS, JR.—*Deutsch. Archiv f. klin. Med.*, B. LXIV.

HIS.—*Semaine Médicale.* 1895, p. 408.

KRAUSE.—*Inaug. Dissert., Göttingen*, 1895.

VON SCHUSTER.—*Deutsch. med. Wochenschrift*, 1896.

ZURHELLE.—*Nothnagel.* XI., I, p. 241.

HAMMER.—*Wiener med. Wochen.*

BELSKI.—*Zeitschr. f. klin. Med.*, B. LVII. Heft 5 and 6.

NAUNYN.—Ueber senile Epilepsie und das Griesinger'sche Symptom der basilar Thrombose.

LOEB.—*Arch. f. die gesammte Physiol.*, G. VII., 5-6, p. 253.

KREHL.—*Spec. Pathol. und Therapie von Nothnagel.* Krehl, Die Erkrankungen des Herzmuskels, etc.

ROMBERG.—*Handb. der prakt. Med. von Ebstein und Schwalbe. Krankheiten der Kreislaufsorgane.*

GERHARDT.—*Lehrb. d. Auscult u. Perc. V. Aufl.*, S., 221.

HERING.—*Pfluger's Archiv*, CVIII., p. 267.

HERING.—*Prager med. Wochenschr.*, 1896, 6.

MORITZ.—*St. Petersburg med. Woch.*, 1897, p. 301.

EDGREN.—*Die Arteriosklerose*, Leipzig, 1898.

HOCHHAUS AND QUINCKE, *Deutsch. Arch. f. klin Med.*, 53, p. 414.

NEUBERGER AND EDINGER.—*Berl. klin. Wochen.*, 1898, 4.

HUCHARD.—*Traite clinique des maladies du cœur et le d'aorta*, 1899.

A. HOFFMAN.—*Zeitschr. f. klin. Med.*, B. XLI., p. 357, 1900, in *Patholog. u. Therapie der Herzneurosen.* Wiesbaden, 1901.

WENCKEBACH.—*Zeitschr. f. klin. Med.*, XXXIX., p. 302, 1900.

A. JACQUET.—*Deutsch. Archiv f. klin. Med.,* B. LXXII.

LUCE.—*Deutsch. Archiv f. klin. Med.,* 1902.

BREUNIG.—*Arch. f. Physiol.,* 1904; Suppl. I.

RETZER.—*Arch. f. Physiol.,* 1904, I, p. 278.

ERLANGER.—*Centralbl. f. Physiol.,* 1905, XIX., p. 9.

TAWARA.—*Centralbl. f. Physiol.,* 1905, XIX., 70, 298.

HERING.—*Archiv f. die gesammte Physiol.,* 1905, G. VII., p. 97.

KRONEEKES.—*Compt. rend. de l'Académie des Sciences.* Paris, 1905, CXLI., p. 529.

ASCHOFF.—*Münchener med. Wochen.,* Sept. 26, 1905.

FINCKELBURG.—*Deutsch. Arch. f. klin. Med.,* Apr. 6, 1905.

EINTHOVEN.—*Arch. Neerl. d. se. exactes,* Harlem, 1906, ser. 2, toma XI.

LICHTHEIM.—*Deutsch. Archiv f. klin. Med.,* B. LXXXV. Heft 3 and 4.

JOACHIM.—*Deutsch. Archiv f. klin. Med.,* B. LXXXV. Heft 3 and 4.

BROUARDEL.—*Arch. d. med. Exper.,* 1906, XVIII., 230.

SCHMOLL.—*Deut. Arch. f. klin. Med.,* 1906, L. XXXVII., p. 554.

BROUARDEL AND VILLARET.—Arch. de med. experim. et d'anat. pathol. Mars, 1906.

ENRIQUEZ AND AMBARD.—*Semaine Médicale,* Jan. 1907, Vol. XXVII. No. 4, pp. 40-42.

RIEGEL.—*Zeitschr. f. klin. Med.,* B. XVII., Heft 3 and 4.

VI.

TACHYCARDIA

FOREIGN BIBLIOGRAPHY

Nothnagel.—*Wein med. Blätter,* 1887.

Pitres.—*Archiv. clin. de Bordeaux,* 1894.

A. Hoffmann.—*Die paroxysmale Tachycardie,* Wiesbaden, 1900.

Derselbe.—*Pathologie und Therapie des Herzneurosen,* Wiesbaden, 1901.

Rose.—*Berl. klin. Wochenschr.,* 1901, No. 27 u. 28.

Derselbe.—*Deutsch. Arch. f. klin. Med.,* Bd. 78, 1903, S. 39 ff.

Wenckebach.—*Die Arythmie.,* Leipsic, 1903.

Schlesinger and Pal.—*Centralbl. f. innere Medizin,* 1903.

Derselbe.—*Zeitschrift f. klin. Med.,* Bd. 53, 1904, S. 206 ff.

Krehl.—*Pathologische Physiologie,* 3 Aufl., 1904.

Muller.—*Multiple Sclerose.,* Jena, 1904.

Lommel.—*Deutsch. Arch. f. klin. Med.,* Bd., 82, 1905, S. 495 ff.

Gerhardt.—*Deutsch. Arch. f. klin Med.,* Bd. 82, 1905, S. 509 ff.

Kraus.—*Deutsch. med. Wochenschr.,* 1905, No. 1, 2 u. 3.

Vollhard.—*Münch med. Wochenschr.,* 1905.

Hirschberg (Paris).—*Neurolog. Zentralbl.,* 1906, No. 1, S. 21.
Hoesslin.—*Morbus Basedowii.*
Schlesinger.—*Sammlung klin. Vorträge,* 433.
Rihl.—*Deutsch. med. Wochenschr.,* Berlin and Leipsic.
Hering.—*Verhandl. d. Kongr. f. innere Med.,* 1906.
Engelmann.—*cf. Wenckebach, l. c.*
Ders.—*Archiv. f. d. ges. Physiol.,* Vol. 62.
Engelmann.—*Pflüger's Archiv.,* Vol. LXII.
His.—*Ztschr. f. exper. Path.,* Vol. II.
Gerhardt.—*Archiv. f. klin. Med.,* Vol. LXXXI.
Pan.—*Ztschr. f. exper. Path.,* Vol. II.
Herz.—*Deutsche med. Wochenschr.*
A. Hoffmann.—*Ztschr. f. klin. Med.,* Vol. 53.
Reinold.—*Ztschr. f. klin. Med.,* Vol. LIX.
Hoffmann.—*Archiv. f. d. ges. Physiol.,* Vol. 84.

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